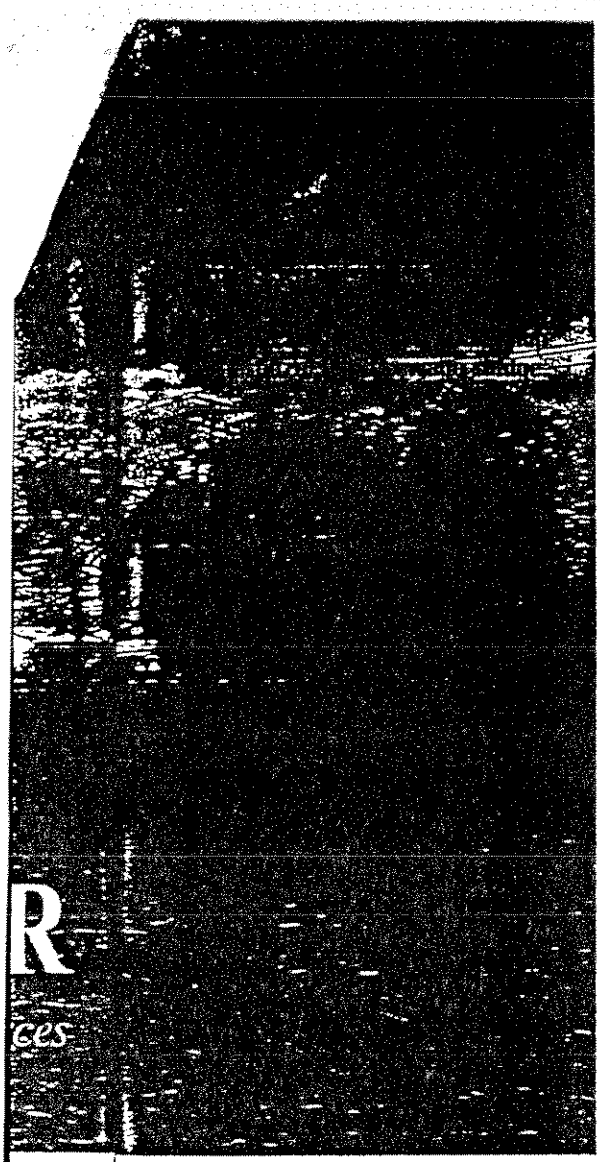

PHOTO: DENNIS GARNICA

mium from the soil. "It appears feeding [tobacco] plants causes a direct increase in cadmium levels in the animal. Spreading them back on the fields increases the soil's overall cadmium level, which I believe leads to more cadmium available to the forage or other crops grown on that soil," Swerczek says.

He and Smith agree that overall use of fertilizers and sewage sludges that contain cadmium, even at trace amounts, is slowly increasing cadmium in fields, and the cadmium level in plants grown in those fields.

On average, U.S. soils contain 0.3 ppm cadmium, though levels can reach 80 ppm to 100 ppm in areas with naturally rich cadmium deposits.

Rock-based fertilizers such as phosphate and zinc often contain some cadmium, which is brought up when the ore is mined. Phosphates can have parts-per-million cadmium levels in the single digits, or reach as high as 220 ppm from ores mined in Idaho and Wyoming. Some zinc fertilizer sources, produced from industrial wastes and byproducts have even higher levels.

Tests in the United States, Canada, Australia and across Europe show forages, potatoes, grains and most other crops can all pull cadmium from the soil. Actual cadmium uptake varies greatly depending on the plant and on the soil's organic matter and pH. Cadmium uptake by plants increases as soil pH falls below 6.5.

Canada, Australia and many European countries have regulations on maximum allowable cadmium levels in fertilizers and other products applied on land. To date, the only U.S. standards for cadmium, as well as other hazardous heavy metals, are based on worker protection issues aimed at levels tolerable for fertilizer plant workers. There are no regulations to address long-term concerns, nor is there any testing or labeling of fertilizers that contain heavy metals.

U.S. fertilizers are regulated by each state, not federal agencies. State regulators have historically focused on making sure fertilizers contained the amount of plant nutrients advertised. There has been little examination of what might be in a fertilizer besides the advertised nutrients.

In response to concerns over hazardous materials in fertilizers, the American Association of Plant Food Control Officials (AAPFCO) in February recommended that states adopt interim standards based on Canadian regulations for lead, arsenic and cadmium levels. These regulations require fertilizer manufacturers to test products for cadmium and other hazardous materials, then create directions for use so users will not apply more cadmium than is deemed safe for their soils. Such safe levels will be up to each state to develop.

Some fertilizer manufacturers disagree with standards based on Canadian levels. "We're for interim standards to be used while the industry [and Environmental Protection Agency (EPA)] begins testing procedures to see what heavy metal levels are truly safe," says Ron Phillips, spokesman for The Fertilizer Institute (TFI). "But we want interim standards that are based on some form of risk assessment, not just numbers created by professional judgements, as the Canadian standards are."

One TFI study showed that applying the Canadian standards to a hypothetical application of a phosphate fertilizer would allow it to contain only 171.5 ppm cadmium.

TFI proposes using EPA sewage sludge standards for heavy metals as an interim fix. Sludge standards were based on scientific studies of the potential for heavy metals reaching human diets. The same TFI study showed that by using sewage sludge standards, the hypothetical fertilizer application could have 751.4 ppm cadmium. Essentially all U.S. phosphate fertilizers would clear a sludge-based regulatory bar, Phillips says.

George Latimer, state chemist in charge of Texas fertilizer inspection and a leader in the AAPFCO push to adopt Canadian-based standards, says sludge standards, which are based on a risk assessment for organic sources, aren't necessarily applicable to inorganic fertilizers like phosphate rock. "With our proposed standards, if a company has a product with high cadmium levels, they will simply have to blend those sources or otherwise direct the purchaser to apply that fertilizer at a rate that won't exceed cadmium levels," Latimer says.

Rufus Chaney, agronomist with USDA's Environmental Chemistry Lab, is a leading U.S. expert on cadmium and other heavy metals in the soil and in fertilizers. Chaney says his research—much of which centered on aiding EPA in developing the sewage sludge standards—has never found brain or other problems with animals fed low levels of cadmium, as Tom Swerczek has. Yet he agrees it is time to be concerned about cadmium levels added to the environment, including taking a hard look at what is applied to the land each year.

> "If we continue with the same practices for another 20 years or so, we might have problems"

"If we're concerned about cadmium, and I think we should be, we should move away from these Western sources of high-cadmium phosphate fertilizers. If we're using micronutrients such as zinc that contain higher levels of cadmium than other, readily available sources, then we ought to quit using them as well.

"This isn't a situation where anyone is going to die tomorrow," Chaney says. "But if we continue with the same practices for another 20 years or so, then we might have problems." *FJ*

Cadmium

Introduction

Cadmium is an element that occurs naturally in the earth's crust. Pure cadmium is a soft, silver-white metal; however cadmium is not usually found in the environment as a metal. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide). These compounds are solids that may dissolve in water but do not evaporate or disappear from the environment. All soils and rocks, including coal and mineral fertilizers, have some cadmium in them. Cadmium is often found as part of small particles present in air. You cannot tell by smell or taste that cadmium is present in air or water, because it does not have any definite odor or taste.

Most cadmium used in this country is extracted during the production of other metals such as zinc, lead, or copper. Cadmium has many uses in industry and consumer products, mainly batteries, pigments, metal coatings, and plastics.

Fate & Transport

Cadmium can enter the environment in several ways. It can enter the air from the burning of coal and household waste, and metal mining and refining processes. It can enter water from disposal of waste water from households or industries. Fertilizers often have some cadmium in them and fertilizer use causes cadmium to enter the soil. Spills and leaks from hazardous waste sites can also cause cadmium to enter soil or water. Cadmium attached to small particles may get into the air and travel a long way before coming down to earth as dust or in rain or snow. Cadmium does not break down in the environment but can change into different forms. Most cadmium stays where it enters the environment for a long time. Some of the cadmium that enters water will bind to soil but some will remain in the water. Cadmium in soil can enter water or be taken up by plants. Fish, plants, and animals take up cadmium from the environment.

Exposure Pathways

Food and cigarette smoke are the largest potential sources of cadmium exposure for members of the general population. Average cadmium levels in U.S. foods range from 2 to 40 parts of cadmium per billion parts of food (ppb). Average cadmium levels in cigarettes range from 1,000 to 3,000 ppb. Air levels in U.S. cities are low, ranging from 5 to 40 nanograms per cubic meter. The level of cadmium in most drinking water supplies is less than 1 ppb. In the United States, the average person eats food with about 30 micrograms (ug) of cadmium in it each day. About 1 to 3 ug per day of cadmium is absorbed from food, and smokers absorb an additional 1 to 3 ug per day from cigarettes. Smoke from other people's cigarettes probably does not cause nonsmokers to take in much cadmium. Cadmium is found at hazardous waste sites at average concentrations of about 4 ppb in soil and 5 ppb in water. Workers can be exposed to cadmium in air from making cadmium products such as batteries or paints. Workers can also be exposed from working with metal by soldering or welding. Each year almost 90,000 workers are exposed to cadmium in the United States.

News releases

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The Growers Solution

SPRING 2001

VOLUME 14 ISSUE 2

On The Road Again in 2001

HOPE TO SEE YOU

Growers Nutritional Solutions is scheduled to set up and staff booths at the following upcoming farm shows and conventions this spring, summer, and fall. It's a great time to stop in and review your fertilization program, hear about new developments at Growers, or just chat with the folks who make it all happen - your friends and neighbors.

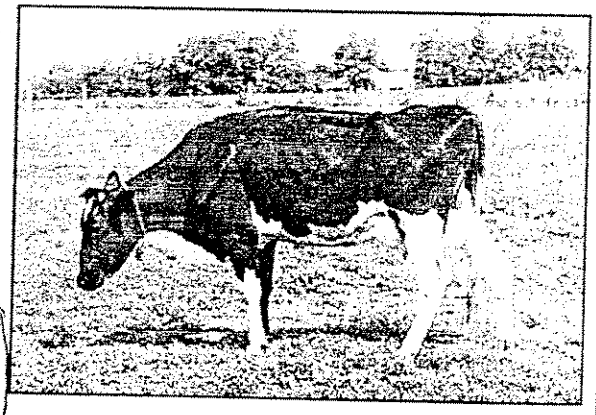
March 29-31	Pennsylvania Beef Exp State College, PA
July 10-12	Michigan Ag Expo E. Lansing, MI
August 7-8	Farm Focus Van Wert, OH
August 7-9	Empire Farm Days Seneca Falls, NY
August 7-9	FarmFest Redwood Co., MN
August 14-16	Ag Progress Days Rock Springs, PA
September 18-20	Wisc. Farm Progress Days Janesville, WI
September 18-20	Ohio Farm Science Review London, OH
September 25-27	Farm Progress Show Lafayette, IN
October 4-6	Carolina Farm Show Kinston, NC

Annual Meeting: Special Report

Solution to Cadmium Toxicity

Cadmium is considered one of the most dangerous occupational and environmental poisons for humans. For years numerous scientific reports, primarily from Europe and Japan, have indicated that chronic environmental cadmium exposure is potentially very detrimental to human health. However, there has been very limited information on the effect of chronic environmental cadmium toxicosis in plants and livestock. It wasn't until we became aware of the work of Dr.T.W. Swerczek, DVM, Ph.D., which was featured in "Heavy Metals in Fertilizers" in the *Farm Journal's* 1998, April issue, did we at Growers Nutritional Solutions and likely many others in the industry, realize the significance of this widespread environmental toxicosis which is affecting crops and livestock.

Dr. Swerczek was an invited guest speaker at our annual meeting 3 years ago. At that time he discussed his research on the effect of chronic environmental cadmium toxicosis in plants and animals. He reported that he suspected zinc sulfate as one of the several sources of cadmium toxicosis for feedstuffs and fertilizers. The Environmental Protection Agency recently confirmed Dr. Swerczek's findings by reporting that several thousand tons of zinc sulfate, highly contaminated with cadmium, have been imported from China over the past several years. This contaminated zinc sulfate has been used in feedstuffs and fertilizers throughout the U.S. Other significant sources of cadmium are zinc containing minerals that are not purified, rock phosphates, industrial wastes in sewage sludge and polluted air from the burning of fossil fuels.



Three year-old Hostein heifer showing signs of poor body condition, elevated back, thin narrow neck and rough, rust color hair coat that failed to shed. Dr. Swerczek believes these symptoms are related to cadmium toxicity.

Dr. Swerczek also discussed with us 3 years ago that many feedstuffs, forages and soils have excessive amounts of iron. He pointed out that excessive iron acts like a double-edged sword in that it will not only tie up essential minerals, like copper, which is essential for the immune system, but excessive iron may aggravate gram negative bacterial infections like E.coli and Salmonella. Excessive iron in rations along with cadmium contamination will severely deplete animals of copper making them susceptible to a host of opportunistic diseases. Fortunately, major feed companies are now reporting that they will no longer fortify mineral supplements with iron, primarily iron oxide, as excess amounts will tie up copper.

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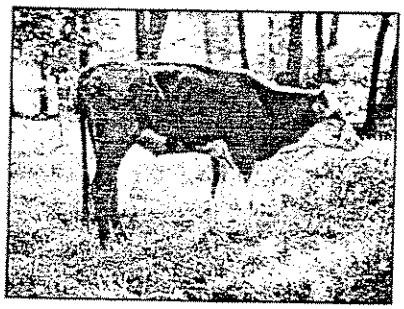
Iron is an essential nutrient in small amounts, but may be toxic in excessive amounts. Like iron, cadmium, which is one of the most toxic of all heavy metals is found in trace amounts in the environment and in most soils as a contaminant of zinc. But, unlike iron, cadmium is not an essential nutrient, and in minute amounts may induce toxicosis.

RESEARCH RESULTS UPDATE

Again this year, Dr. Swerczek was an invited guest speaker at our Growers Nutritional Solutions annual meeting and he gave us an update on his research on chronic environmental cadmium toxicosis in plants and animals. He reiterated that cadmium toxicosis is the most overlooked and the most difficult heavy metal toxicosis to diagnose because of the nature the



Two year-old Semmental heifer showing signs of chronic wasting. Note the elevated back, emaciation, rough hair coat, submandibular edema, and staining of the tail from fecal matter due to watery diarrhea. These are symptoms Dr. Swerczek believes are related to cadmium toxicity.

metal. Cadmium is very similar in structure to essential nutritional metal ions, but it is not an essential nutrient, even in minute amounts. Chronic exposure to this very toxic heavy metal will produce an indirect form of toxicosis as it has a negative influence on hundreds of enzymatic systems of cells as it will substitute for essential metal ions, primarily zinc and copper in metalloenzymes.* Also, cadmium has a strong affinity for biological structures containing SH groups.* As a result, the indirect effect on multiple cellular and enzymatic systems will produce many negative effects on the health of plants and animals. The clinical effects of poor health are seen as deficiencies of essential metal ions like copper and zinc, and perhaps others like selenium and magnesium, which are induced deficiencies caused by the cadmium. In most cases, all of these essential minerals are adequate in the diet, but are being inhibited by or substituted for in critical cell and enzymatic systems by cadmium. Probably the most significant negative effect of cadmium toxicosis is on the immune system where it produces a severe immune suppression. The end result of cadmium toxicosis is poor health and the induced susceptibility to a multitude of metabolic and hormonal disorders and numerous opportunistic diseases of plants and livestock.

Because cadmium toxicosis produces toxic effects indirectly, tissue levels of cadmium by themselves are meaningless as an indicator of cadmium toxicosis. Rather, an understanding of the indirect effects of cadmium toxicosis, primarily induced deficiencies of essential metal ions in important cellular and enzymatic systems, and induced immune suppression, have to be taken into account. Unless the overall clinical syndrome and the indirect effects of cadmium toxicosis are understood, the massive destructive effects of cadmium toxicosis in plants and animals will continue to go unrecognized.

INADEQUATE ANALYTIC PROCEDURES

Another reason that cadmium toxicosis has been overlooked and misdiagnosed is because the analytic procedures for cadmium have been inadequate. Cadmium is very difficult to analyze for and only a few certified laboratories are capable of accurately analyzing for cadmium. In testing for cadmium, other minerals in the sample matrix, primarily phosphates and organic materials in the sample being tested may interfere with cadmium which results in false negative values.

Because cadmium is toxic in very small amounts it is essential that analytical procedures be performed accurately with adequately controlled procedures. The most accurate method to test for cadmium is neutron activation analysis. Other procedures may be used but they are not as sensitive or accurate.

The willow tree grown in zinc and cadmium contaminated soils is concentrating cadmium, and is causing toxicosis to many forms of wildlife.

With all analytical procedures, during the processing and testing, cadmium is broken down to the elemental form, which is then quantified. It is very important that any test results be interpreted with caution. Most testing procedures are very limited and they have to be interpreted in regard to the form in which the cadmium is found. For example, cadmium found in a sample of calcium is likely in an inorganic form and may be relatively non-toxic to plants and animals, as calcium will protect against the uptake of cadmium. On the other hand, the same amount of cadmium found in a sample of zinc or phosphates may be very significant, as it may be highly toxic to plants and animals, especially when there is a calcium deficiency in soils and in livestock rations.

Calcium will protect against the uptake of cadmium.

When cadmium is coupled or chelated by organic materials like amino acids, peptides, proteins and carbohydrates, it may be extremely toxic in minute amounts and readily available to plants and animals. Dr. Swerczek feels that bioactivation of cadmium, either naturally by microorganisms and plants or unintentionally during processing of feedstuffs and fertilizers, is the most significant and unrecognized form of cadmium toxicosis in plants and animals today. Dr. Swerczek pointed out that the tobacco plant may concentrate cadmium, but with the currently available analytical techniques for cadmium, only the elemental form of cadmium is quantified. The very toxic bioactivated organic forms that these plants likely contain, most all of which are unknown, are not determined by currently available testing procedures.

THEORY SUPPORTED

To help support his theory on the bioactivation of cadmium, he cited recent research by scientists working in the Rocky Mountain area of Colorado. This work showed that the willow tree grown in zinc and cadmium contaminated soils is concentrating cadmium, and is causing toxicosis to many forms of wildlife in the area, including birds, beaver, deer and elk that feed on these trees. These workers concluded that cadmium poisoning may be more widespread than shown in their study. Like Dr. Swerczek, these workers state that their results suggest that cadmium poisoning may be more common among natural populations of vertebrates than has been appreciated to date and that cadmium toxicity may often go undetected or unrecognized. They also state that even trace quantities of cadmium not only influence the physiology and health of individual organisms, but also the demographics and the distribution of species.

Similar to the findings in the Colorado Rocky Mountains, the tobacco plant, like willow trees, concentrates cadmium. If either of these plants is grown on soils fertilized with nutrients contaminated with cadmium, these plants will uptake and concentrate cadmium. Dr. Swerczek and other veterinarians have observed that cattle

*__metalloenzyme:__ *An enzyme is a protein with catalytic properties, that is, it increases the speed of a chemical reaction without being consumed or irreversibly altered by the reaction process. A metalloenzyme is an enzyme (or protein) that is associated with a metal cation (such as calcium or zinc). In many biological processes the enzyme can only function if the enzyme is complexed with a particular metal cation. This reveals that the process (the lock) will not function without the proper metalloenzyme (the key).*

SH groups *are what are termed sulfhydryl groups and they are usually associated with enzymes and allow enzymes to catalyze biological processes in the same lock and key relationship as with metalloenzymes. The only difference is that sulfhydryl groups control the enzyme through pH changes rather than by specific metal cations.*

that graze grass fertilized with tobacco stalks from plants that have been fertilized with phosphates, a known source of cadmium, have a high incidence of metabolic disorders with clinical signs of tetany and paresis.* It is Dr. Swerczek's theory that the cadmium is leaching out of the tobacco plants and is being absorbed

Cattle that graze grass fertilized with tobacco stalks from plants that have been fertilized with phosphates have a high incidence of metabolic disorders.

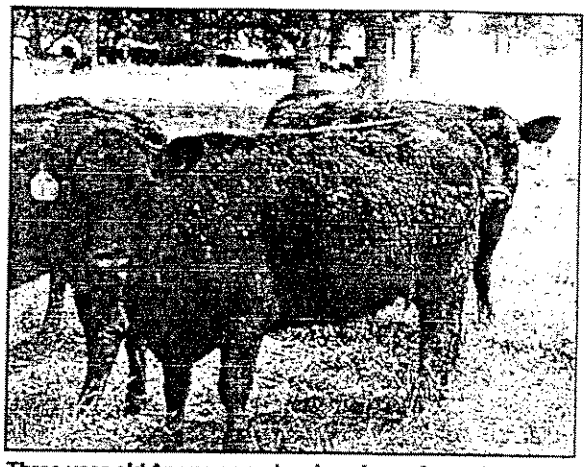
by rapidly growing grass and a biologically active form of cadmium is readily available to cattle through the grass. The cadmium replaces essential metal ions at the cellular level, especially calcium and magnesium. In many cases there is a hypocalcemia* which further enhances the absorption of cadmium. Magnesium is usually available, in many cases in excessive amounts, but like copper, there may be an induced deficiency of magnesium at the cellular level.

METABOLIC DISORDERS TRACED

Dr. Swerczek has observed that, if cattle graze pastures that are heavily fertilized, seemingly, they are more susceptible to metabolic disorders like grass tetany. Similarly, he has also observed that if fescue grass is heavily fertilized, clinical signs of fescue toxicosis are exacerbated. Interestingly, Dr. Swerczek indicated that many of the clinical signs of fescue toxicosis are identical to those seen in cattle suffering from clinical signs consistent with chronic environmental cadmium toxicosis in that both have clinical signs of an induced copper deficiency. He now feels that the fescue plant may be similar to willow trees and tobacco plants, and may be also concentrating cadmium. The fungal endophyte within the fescue plant may be bioactivating the cadmium, working synergistically to make both the endophyte and cadmium more available and toxic to the animal. Dr. Swerczek suggested that since the fescue plant is native to mountainous areas and thrives on poor high mineral soils where other grasses fail, it may be concentrating cadmium similar to willow trees in the Rocky Mountains.

The fescue plant may be similar to willow trees and tobacco plants, and may be also concentrating cadmium.

He has support for this theory with the work he has observed on the Bill Gess farm, near Lexington, Kentucky, where cattle not only have had clinical signs of grass tetany, but also had severe clinical signs of fescue toxicosis. The forced feeding of both magnesium and copper did not prevent clinical signs of either syndrome. This farm had fertilized pastures that had a considerable amount of endophyte infected fescue grass, with tobacco stalks and phosphates fertilizers.



Three year-old Angus cow showing signs of poor body condition: rough, long rust color hair that is not shedding. All cows in herd showing same clinical signs believed to be signs of cadmium toxicity, and were grazing a mixed grass pasture with fescue grass.

SOLUTION OFFERED

At Dr. Swerczek's recommendations, the farm stopped spreading tobacco stalks on the pastures and the pastures were not fertilized with nutrients likely to contain cadmium. Also, the cattle were not supplemented with nutrients likely to contain excessive amounts of cadmium.

FARM'S PROGRAM

Bill Gess outlined and followed a program with the help of the farm's veterinarian in an attempt to overcome clinical signs of chronic environmental cadmium toxicosis on the farm. For two years, the pastures were not fertilized with tobacco stalks or fertilizers likely to contain excess cadmium, and the cattle were fed non-fortified salt and calcium carbonate with added copper sulfate. On the third year, calcium and Growers Nutritional Solutions were applied to the pastures several times during the growing season. In addition, the cattle had access to free choice Growers Nutritional Solutions.

Remarkably, the grass was lush with this practice and all signs of grass tetany and fescue toxicosis disappeared in a large herd of beef cattle. The cattle on the farm are now healthier, cows have high calving rates, calves are very vigorous at birth and the weaning weights are higher in the calves. What is even more remarkable, the farm has the same amount of endophyte infected fescue, and clinical signs of fescue toxicosis and grass tetany have disappeared in the herd.

Observations support the theory that cadmium may be involved with both grass tetany and fescue toxicosis.

QUESTIONS & POTENTIAL ANSWERS

The observations on this farm support Dr. Swerczek's theory that cadmium may be involved with both grass tetany and fescue toxicosis. Currently, the mechanisms of action for the cause of both syndromes are poorly understood. Dr. Swerczek's observations are extremely interesting and merit further study as grass tetany and fescue toxicosis are both very costly diseases of the cattle industry.

He cited another precedent for his theory that cadmium may be bioactivated by microorganisms and plants. The Japanese scientists have shown years ago that inorganic forms of mercury, another similar very toxic heavy metal, are being bioactivated to very toxic organic forms, primarily methylmercury, by microorganisms and fish. For this reason he feels that currently available analytical procedures for cadmium are meaningless in terms of levels found in specimens tested. To determine the significance of cadmium in a sample, the biological form and bioactivity of cadmium is important.

Dr. Swerczek provided us with numerous case histories from several farms from different states that have had severe crop and livestock losses with clinical signs consistent with chronic environmental cadmium toxicosis. He pointed out that in all cases where the source of cadmium contamination was identified and removed, crops and livestock have made dramatic recovery towards better health. Unfortunately, these improvements will not occur immediately as cadmium will remain in the soil until plants remove it and in exposed animals it will remain in the body for months and years. The most dramatic results are seen when new crops and new animals are not exposed to significant amounts of this very toxic heavy metal.

Dr. Swerczek pointed out that soils on farms

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*paresis: *partial paralysis affecting muscular motion but not sensation.*

hypocalcemia (milk fever): *a metabolic disorder in which calcium control mechanisms fail to maintain normal blood plasma calcium concentrations at the onset of lactation. In other words blood calcium concentration decline in nearly all cows at calving as a result of the sudden onset of milk production. In some cows, blood calcium falls to the point where it no longer will support nerve and muscle function and the cow goes down with milk fever.*

with an adequate amount of calcium, and farming programs that utilize calcium and purified fertilizers have very healthy crops. The soils on these farms are healthier and farmers are reporting the return of numerous earthworms.

Farms with adequate calcium or that utilize calcium and purified fertilizers have very healthy crops.

Cadmium is not only very detrimental to plants and animals, it is also very destructive to beneficial microorganisms and earthworms that are essential for optimal soil health. Even more remarkable than the health of the plants, crops and vegetables grown on these soils is the health of the livestock that are being fed these high quality healthier crops and forages. These animals will not only consume less feed, but they perform better and are nearly free of metabolic disorders, induced mineral deficiencies and opportunistic diseases that are plaguing the livestock industry today.

Biographical Information

Dr.T.W. Swerczek, DVM, Ph.D received his B.S. and DVM degrees from Kansas State University in 1964, a M.S. degree in Nutritional Pathology in 1966 and a Ph.D. in Comparative Pathology in 1969 from the University of Connecticut.

Dr. Swerczek's research is in the area of Nutritional Pathology and The Interrelationship between Nutrition and Infectious Diseases, and the effect of environmental plant toxins on induced essential mineral deficiencies and immune suppression of livestock.

As a Veterinary Pathologist, Dr. Swerczek has performed several thousand necropsies of livestock of all classes, including fetuses, neonates and adult animals. He has extensive experience in working with veterinarians, livestock owners, farm managers on nutrition and its effect on infectious diseases of livestock.

Dr. Swerczek is a native of Nebraska with a farm background. He consults with his brother Steve who operates the family's farm in Nebraska where their cow-calf operations are raised on native grasslands. Corn, soybeans and alfalfa are grown on land fertilized with purified nutrients. The feedlot cattle are raised on rations free of antibiotics and hormones.

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Cadmium analyses by various independent testing laboratories on Growers Nutritional Solutions show very low levels compared to other fertility and mineral supplements.

Dr. Tom Swerczek (right, wearing camera) and Wallace Garrett (next to Dr. Swerczek) discuss an examination of Garrett's cattle (review *The Growers Solution*, Fall, 1999).

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FIRST CLASS

Inside:
A discussion concerning one of the most dangerous occupational & environmental poisons

SPRING 2001

Letters to the Editor

Chronic environmental cadmium toxicosis in horses and cattle

Generalized osteochondrosis has been observed sporadically in suckling foals on central Kentucky horse farms for over 25 years.[1-3] In the summer of 1997, a Kentucky horse farm had an outbreak of osteochondrosis in suckling foals. This farm had 20 Thoroughbred mares with suckling foals and 12 yearling foals. There were also 2 Holstein steers that were 2 years old and 1 aged beef cow.

The 3 cattle had grazed the same lush bluegrass pastures as the affected foals. The beef cow had a long, rough coat, was stunted and underweight, and had obvious bone and joint disease in all limbs. The 2 Holstein steers were also underweight, had long, rough coats and their black hair had faded to rust. Several suckling foals and yearlings also had long, rough coats that failed to shed properly.

During the investigation of this outbreak, 3 foals developed clinical signs of osteochondrosis. One 4-month-old foal was severely affected and was euthanized. The clinical diagnosis was confirmed by postmortem examination. The articular cartilage in this foal was either separated or separating from the underlying bone in all joints of the lower portion of the limbs. Toxicologic examination of the foal's liver and kidney revealed low concentrations of copper and cadmium and high concentrations of zinc.

Postmortem findings of this foal were remarkably similar to those reported for suckling foals exposed to environmental zinc and cadmium from a smelter plant.[4] While investigating this recent outbreak of osteochondrosis, it was learned that within the past 2 years some paddock fences had been hand scraped and pressure washed with water to remove several layers of old white paint. The removed paint had, in turn, washed into the pastures. An analysis of the white paint remaining on the board fences revealed high concentrations of zinc contaminated with high concentrations of cadmium.

Cadmium is found in nature as a contaminant of zinc, and both have a high affinity for phosphates.[5] Rock phosphates used in feed and fertilizers generally have high concentrations of cadmium.[5] It is suspected that animals on this farm may have been exposed to several sources of cadmium.

Acute cadmium poisoning has been reported in calves and has caused severe copper deficiency, poor weight gain, emaciation, and death.[6] These findings are remarkably similar to cattle and other livestock in Kentucky suffering from natural severe copper deficiency, although they seemingly have adequate or higher than normal concentrations of copper in their diets. Because cadmium accumulates in the body, it is likely that chronic exposure to low concentrations of this toxic metal may induce a copper deficiency similar to acute cadmium exposure at high concentrations. At necropsy, the concentration of copper in the affected foal was low.

Cadmium may replace copper and zinc at active metabolic sites[7] and in so doing cause severe induced copper deficiency, which is verified when tissues are analyzed at necropsy. Ironically, it is highly probable that cadmium is also causing a severe, induced zinc deficiency at metabolic sites and the enzymologic level, but zinc concentrations are often normal to high in organs analyzed in animals with cadmium toxicosis.[8]

T. W. Swerczek, DVM, PhD
Lexington, Ky

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Cornell University
13-Jul-00

Cadmium Makes Brittle Bones in Rocky Mt. Birds

Library: SCI
Keywords: CADMIUM MINES ROCKY MOUNTAINS COLORADO WILDLIFE ECOLOGY
Description: Toxic cadmium from abandoned mines in Colorado is destroying the bones of a little known bird, the white-tailed ptarmigan, and may be harming other Rocky Mountain wildlife, Cornell University ecologists report in the July 13, 2000, Nature.

FOR RELEASE: 2 p.m. Eastern July 12, 2000

Contact: Roger Segelken
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ITHACA, N.Y. -- Large portions of the Rocky Mountains may not be as pristine as once thought, according to a report by ecologists from Cornell University, the Institute of Ecosystem Studies and the U.S. Geological Survey (USGS) in the July 13 issue of the journal Nature (Vol. 408, No. 6792, pp. 181-183).

Their study, which was funded by the National Geographic Society, focused on abandoned mining districts in Colorado and a little-known species of grouse called the white-tailed ptarmigan (Lagopus leucurus). According to the lead author of the report titled "Cadmium Toxicity Among Wildlife in the Colorado Rocky Mountains," the metal is affecting the ptarmigan in mining areas and may threaten some populations of the bird with extinction. Cadmium from abandoned mines may also affect other wildlife species in the area, including deer, elk, moose, rabbits, beaver and other birds, the researchers predict.

"Even humans in the region may not be immune to the effects of cadmium," said James R. Larison of Cornell and Oregon State universities.

Other authors of the Nature report are Gene E. Likens, director of the Institute of Ecosystem Studies in Millbrook, N.Y., and an adjunct professor of ecology and evolutionary biology at Cornell; John W. Fitzpatrick, professor of ecology and evolutionary biology and director of the Cornell Laboratory of Ornithology; and J.G. Crock, a chemist at the U.S. Geological Survey in Denver. Larison is a doctoral degree candidate at Cornell and a faculty member at Oregon State University in Corvallis.

While the extreme toxicity of cadmium has been established by short-term, lethal-dose experiments, the

ptarmigan study in Colorado's Animas River watershed is the first to show the more subtle, but nonetheless important, result of chronic exposure to excess cadmium in a plant-based diet. Particularly susceptible are animals that forage on willow growing in cadmium-rich soils, say the researchers, who note that willows are especially adept at concentrating the toxic metal in their plant tissues. For the little grouse, cadmium damages kidneys and produces thin, brittle bones that may shorten its life span as well as fragile eggshells that reduce reproductive success.

Cadmium is the silver-white metal used to manufacture rechargeable batteries, produce alloys with other metals and electroplate chrome on auto parts and appliances. It has been mined in the Colorado Rockies for more than a century. Cadmium finds its way into the environment when rainwater washes through the piles of ore tailings at abandoned mine sites.

Most trees and other plants readily take up cadmium from the soil, but willows in particular act as biological pumps to biomagnify, or concentrate, the toxic metal in willow leaves and leaf buds to as much as 100 times the levels found in soil. Those same levels of cadmium were found by the researchers in the crop contents of birds that ate willows.

Once ingested, cadmium can become concentrated in kidney tissue. All the adult birds tested in the central and southwestern mountains of Colorado had elevated kidney-cadmium levels. Toxic levels of cadmium were documented in 44 percent of adult birds, whereas birds living outside the ore belt region of Colorado had near-normal cadmium levels in their bodies. High concentrations of cadmium damage kidney tissue and reduce the birds' ability to process calcium. Among adult birds tested in Colorado, 57 percent had damaged kidneys.

To make matters worse, the mining areas that are rich in cadmium also tend to be calcium poor -- in part because of the high acidity of the water and soils --- so birds have little chance of accumulating the mineral they need for strong bones and robust eggshells. According to Larison, birds with insufficient calcium in their diets are even more likely to accumulate toxic cadmium levels. He discovered the most profound cadmium problems among female grouse, which stay in the mining areas during the winter months when willows are their principal source of sustenance.

"Birds in the winter really get hammered," Larison said. "Their bones fracture easily so they die at a younger age and they don't have enough calcium to build normal eggshells." The average ptarmigan could accumulate toxic kidney-cadmium levels after just 600 days of eating cadmium-rich plants, the ecologists estimated for their Nature report.

"The reason that these cadmium-contaminated populations do not go extinct is that new recruits arrive each year from places with normal cadmium levels to replace those that die off," Larison explained.

In addition to the National Geographic Society, the cadmium study was funded by the Colorado Division of Wildlife, the American Museum of Natural History, the American Ornithologists' Union, Sigma Xi, Cornell University and the Cornell Laboratory of Ornithology.

Related World Wide Web sites: The following sites provide additional information on this news release. Some might not be part of the Cornell University community, and Cornell has no control over their content or availability.

-- National Geographic news: http://www.ngnews.com/

-- Ptarmigan photos: http://osu.orst.edu/dept/ncs/photos/index.html

1: Vet Pathol 1998 Nov;35(6):479-87 Related Articles, Links

Epizootic vacuolar myelinopathy of the central nervous system of bald eagles (Haliaeetus leucocephalus) and American coots (Fulica americana).

Thomas NJ, Meteyer CU, Sileo L.

Biological Resources Division, National Wildlife Health Center, Madison, WI, USA.

Unprecedented mortality occurred in bald eagles (Haliaeetus leucocephalus) at DeGray Lake, Arkansas, during the winters of 1994-1995 and 1996-1997. The first eagles were found dead during November, soon after arrival from fall migration, and deaths continued into January during both episodes. In total, 29 eagles died at or near DeGray Lake in the winter of 1994-1995 and 26 died in the winter of 1996-1997; no eagle mortality was noted during the same months of the intervening winter or in the earlier history of the lake. During the mortality events, sick eagles were observed overflying perches or colliding with rock walls. Signs of incoordination and limb paresis were also observed in American coots (Fulica americana) during the episodes of eagle mortality, but mortality in coots was minimal. No consistent abnormalities were seen on gross necropsy of either species. No microscopic findings in organs other than the central nervous system (CNS) could explain the cause of death. By light microscopy, all 26 eagles examined and 62/77 (81%) coots had striking, diffuse, spongy degeneration of the white matter of the CNS. Vacuolation occurred in all myelinated CNS tissue, including the cerebellar folia and medulla oblongata, but was most prominent in the optic tectum. In the spinal cord, vacuoles were concentrated near the gray matter, and occasional swollen axons were seen. Vacuoles were uniformly present in optic nerves but were not evident in the retina or peripheral or autonomic nerves. Cellular inflammatory response to the lesion was distinctly lacking. Vacuoles were 8-50 microns in diameter and occurred individually, in clusters, or in rows. In sections stained by luxol fast blue/periodic acid-Schiff stain, the vacuoles were delimited and transected by myelin strands. Transmission electron microscopy revealed intramyelinic vacuoles formed in the myelin sheaths by splitting of one or more myelin lamellae at the intraperiodic line. This lesion is characteristic of toxicity from hexachlorophene, triethyltin, bromethalin, isonicotinic acid hydrazide, and certain exotic plant toxins; however, despite exhaustive testing, no etiology

was determined for the DeGray Lake mortality events. This is the first report of vacuolar myelinopathy associated with spontaneous mortality in wild birds.

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Cadmium toxicity threatening wildlife in Rocky Mountains

07-12-00

By Mark Floyd, 541-737-0788
SOURCE: Jim Larison, 541-737-8284

CORVALLIS, Ore. - An alarming number of white-tailed ptarmigan in a large region of the southern Rocky Mountains are suffering from acute cadmium poisoning - an exposure to high concentrations of the extremely toxic trace metal.

A new study published in Nature found that an alarming number of white-tailed ptarmigan are suffering from acute cadmium poisoning.

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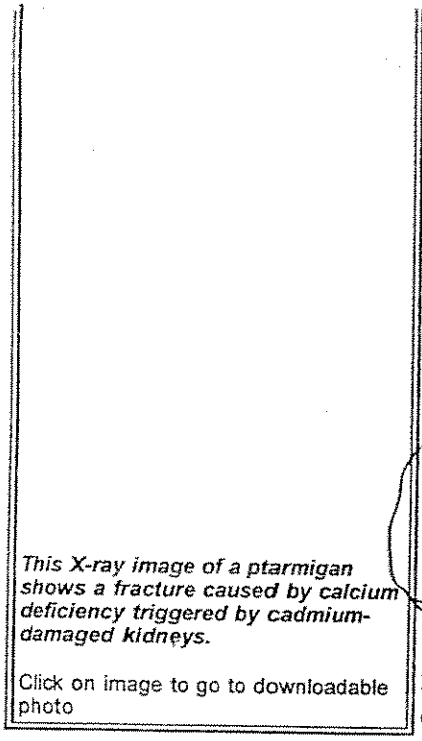
Scientists report Thursday in the journal Nature that 46 percent of the adult birds surveyed in a 10,000-square kilometer area in south-central Colorado were found with cadmium accumulations in their kidneys well above the toxic threshold of 100 parts pe r million.

Cadmium toxicity causes kidney and liver dysfunction, brittle bones, and adversely affects reproduction and survival.

Lead author James R. Larison, an Oregon State University professor and alpine ecologist, said the findings are not unlike those that linked the pesticide DDT to a problem of thin-eggshells in the peregrine falcon three decades ago. The implications of th e toxicity go beyond a single species.

"What we found in our study was that a particular genus of plants - willows - were 'biomagnifying' or concentrating cadmium," Larison said. "They act as biological pumps, increasing the concentrations of cadmium by two orders of magnitude. Birds eat a lo t of willow, especially in the winter when other foods are scarce.

"They aren't the only creatures to eat willow, though," he added. "The possibility exists that deer, elk, moose, snowshoe rabbits, beaver and other animals may face similar problems, just as it is possible that other plants - including some vegetables - may have the same abilities to biomagnify cadmium that willow does."



This X-ray image of a ptarmigan shows a fracture caused by calcium deficiency triggered by cadmium-damaged kidneys.

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Larison said the human health risk from eating ptarmigan likely is small, unless the internal organs are consumed. But, he added, many people eat vegetables grown in the area and these could pose a risk to human health. The former director of Sea Grant Communications at Oregon State University, Larison has spent the past four years at Cornell University pursuing his doctorate in ecology and evolutionary biology. His doctoral study was funded primarily by the National Ge ographic Society. Other authors in the Nature article include Gene Likens, director of the Institute for Ecosystem Studies in Millbrook, N.Y., John Fitzpatrick, director of the Cornell University Laboratory of Ornithology, and J.G. Crock, a chemist with t he U.S. Geological Survey.

The study focused on an expansive section of Colorado stretching from Denver and Fort Collins to Durango known as an "ore belt." Larison, who has returned to the OSU faculty, said abandoned mines throughout this area have "exacerbated the problem."

CWD Belt

Though cadmium is natural to the area, he pointed out, mining tends to mobilize potentially toxic metals. "Cadmium poisoning originally was discovered in Japan, with rice acting as a biomagnifier," Larison said. "Elderly women in particular were affected with severe osteomalacia - a condition not unlike osteoporosis. Trace amounts of cadmium can be found in almost all soils, surface waters and plants, but human activities tend to concentrate it. Mining is one obvious factor, but cadmium also is mobilized by certain industrial and agricultural practices."

Once ingested, cadmium cannot easily be excreted from the body and accumulates, usually in the kidneys and liver. The kidneys are responsible for calcium levels in the blood, Larison said, and when cadmium levels rise and kidneys tubules fail, calcium le vels drop. To compensate, the body "borrows" calcium from bones. In Japan, elderly women eating a diet heavy in cadmium-contaminated rice suffered from severe bone decalcification.

IS THIS CAUSING OSTEOPOROSIS

In Larison's study, 57 percent of the adult ptarmigan had damaged kidneys and their bones contained 8 to 10 percent less calcium.

"We also found a number of birds with bone fractures," he said. "For every one we found, there may have been others that did not survive long enough for us to discover them."

Cadmium toxicity in predators eating ptarmigan is a concern, Larison pointed out, because they likely would eat the internal organs and the cadmium would then accumulate in their bodies as well. Ptarmigan predators include eagles and hawks, as well as fo xes and coyotes.

Though the Nature article focuses on one area in the Rocky Mountains, cadmium poisoning potentially could occur elsewhere, Larison said.

"We happened to look at the effects just on white-tailed ptarmigan eating willows in Colorado," Larison said. "But there are some indications that the conditions for cadmium poisoning are widespread."

-30-

Jim Larison releases a ptarmigan.

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Timeline of Mining In Wisconsin

1658 - Native Americans begin to mine lead ore within the southwestern region of Wisconsin.

1700's - French explorers and Native Americans mine lead on a small scale in southwest Wisconsin.

1776 - Declaration of Independence from Great Britain.

1812-1814 - War of 1812.

1816 - First lead smelter in Wisconsin in LaFayette County.

1825 - First major lead strike in Wisconsin near New Diggings, Hazel Green and Shullsburg.

1827 - William S. Hamilton, son of famous Federalist and first U.S. Secretary of the Treasury Alexander Hamilton, discovers large lead deposit near Wiota.

1828 - Extensive mining begins around Platteville.

1830-1871 - Upper Mississippi Lead District, which includes the southwestern lead region of Wisconsin, is the most important lead producing area of the United States.

1836-1838 - Mexican War.

1848 - Lead mining in Wisconsin begins to decline.

1849 - The Iron Ridge open pit and underground mine begins operation in Dodge County.

1861-1865 - Civil War.

1871 - University of Wisconsin creates a Department of Mining and Metallurgy.

1898 - Spanish-American War.

1908 - Legislature establishes Wisconsin Mining and Trade School in Platteville, forerunner to the current University of Wisconsin - Platteville.

1911 - Wisconsin ranks 3rd among zinc producing states.

1917 - Upper Mississippi District produces 64,000 tons of zinc metal while the United States enters World War I.

1930's - Zinc production declines during the Great Depression.

1940's - Zinc production begins to rise once again as the United States prepares for WW II.

1948-1968 - Wisconsin remains among top 10 zinc production states.

1983 - Wisconsin's last iron mine, the open pit Jackson County Mine, ceases operations and begins reclamation.

1993 - Flambeau Copper Mine in Ladysmith begins operation.

Hazardous Wastes Become Fertilizer

by Duff Wilson

SEATTLE TIMES - When you're mayor of a town the size of Quincy, Wash., you hear just about everything. So it was only natural that Patty Martin would catch some farmers in her Central Washington hamlet wondering aloud why their wheat yields were lousy, their corn crops thin, their cows sickly. Some blamed the weather. Some blamed themselves. But only after Mayor Martin led them in weeks of investigation did they identify a possible new culprit: fertilizer. They don't have proof that the stuff they put on their land to feed it actually was killing it. But they discovered
something they found shocking and that they think other American farmers and consumers ought
to know: Manufacturing industries are disposing of hazardous wastes by turning them into fertilizer to spread around farms. And they're doing it legally.

"It's really unbelievablewhat's happening, but it's true," Martin said. "They just call dangerous waste a product, and it's no longer a dangerous waste. It's a fertilizer." Across the Columbia River basin in Moxee City is visual testimony to Martin's assertion. A dark powder from two Oregon steel mills is
poured from rail cars into the top of silos attached to Bay Zinc Co. under a federal permit to store hazardous waste. The powder, a toxic byproduct of the steel-making process, is taken out of the bottom of the silos as a raw material for fertilizer. "When it goes into our silo, it's a hazardous waste," said Bay Zinc President Dick Camp. "When it comes out of the silo, it's no longer regulated. The exact same material. Don't ask me why. That's the wisdom of the EPA."

What's happening in Washington is happening around the United States. The use of industrial toxic waste as a fertilizer ingredient is a growing national phenomenon, an investigation by The Seattle Times has found. The Times found examples of wastes laden with heavy metals being
recycled into fertilizer to be spread across crop fields. Legally. In Gore, Okla., a uranium-processing plant is getting rid of low-level radioactive waste by licensing it as a liquid fertilizer and spraying it over 9,000 acres of grazing land. In Tifton County, Ga., more than 1,000 acres of peanut crops were wiped out by a brew of hazardous waste and limestone sold to unsuspecting farmers. And in Camas, Clark County, highly corrosive, lead-laced waste from a pulp mill is hauled to Southwest Washington farms and spread over crops grown for livestock consumption.

Recycling said to have benefitsAny material that has fertilizing qualities can be labeled and used as a fertilizer, even if it contains dangerous chemicals and heavy metals. The wastes come from iron, zinc and aluminum smelting, mining, cement kilns, the burning of medical and municipal wastes, wood-product slurries and a variety of other heavy industries.

Federal and state governments encourage the practice in the name of recycling and, in fact, it has some benefits: Recycling waste as fertilizer saves companies money and conserves precious space in hazardous-waste landfills. And, mixed and handled correctly, the material can help crops grow. "It's a situation where we are facing an overabundance of these materials in

landfills and, of course, landfills are getting full," said Ali Kashani, who directs fertilizer regulation in Washington state. "So they (waste producers) are constantly looking for ways to recycle when they have beneficial materials."

The problem is that the "beneficial materials" in industrial waste, such as nitrogen and magnesium to help crops grow, often are accompanied by dangerous heavy metals such as cadmium and lead. "Nowhere in the country has a law that says if certain levels of heavy metals are exceeded, it can't be a fertilizer," Kashani said. "That would be nice to have." Instead, officials rely on fertilizer producers to document that their products are safe, and never check back for toxic components. There is not even a requirement that toxics be listed on ingredient labels. The Times also found that: --
There is no national regulation of fertilizers in this country, unlike many other industrialized nations. The laws in most states, including Washington, are far from stringent. The lack of national regulation makes it virtually impossible to measure the volume of fertilizers produced by recycling hazardous wastes. -- Some industries dispose of tons of toxic waste by giving it free to fertilizer manufacturers, or even paying them to take it. --

One major producer, Monsanto, has stopped recycling waste into fertilizer on its own because of concerns about health and liability. For years, it sold 6,000 tons a year of ashy, black waste from its Soda Springs, Idaho, phosphorus plant to nearby fertilizer companies. The waste contained cadmium, a heavy metal that studies show can cause cancer, kidney disease, neurological dysfunction, diminished fertility, immune-system changes and birth defects at certain levels of consumption. Company scientists are trying to determine whether the material is safe to be used as fertilizer, even though the federal government allows it. "What really is a concern is product liability," said Robert Geddes, a Monsanto official and Idaho state senator.

"Is somebody going to sue Monsanto because we allowed it to be made as a fertilizer?" --
Among the substances found in some recycled fertilizers are cadmium, lead, arsenic, radionuclides and dioxins, at levels some scientists say may pose a threat to human health. Although the health effects are widely disputed, there is undisputed evidence the substances enter plant roots. Just as there are no conclusive data to prove a danger, there are none to prove the safety of the practice. In other nations, including Canada, that lack of certainty has led to strict regulation. There, the approach is to limit toxic wastes in fertilizer until the practice is proven safe. Here, the approach is to allow it until it's proven unsafe.

Although experts disagree as to whether these fertilizers are a health threat, most say further study is needed. Yet, little is under way. Few farmers, and probably even fewer consumers, know about the practice. "This is a definite problem," said Richard Loeppert, a soil scientist at Texas A&M University and author of several published papers on toxic elements in fertilizers. "The public needs to know." Some remember the Alar scarePatty Martin is not a popular politician in parts of Grant County these days. Since she began raising the alarm about the use of toxic waste as fertilizer, she has been threatened with a lawsuit by a local farmer, been verbally attacked in town meetings and seen the City Council - led by a son-in-law of the local manager of the Cenex fertilizer company - pressure her to shut up or quit.

Many farmers in and around Quincy, a town of 4,030, say they're doing very well, thank you, with the fertilizer and the help and advice they've received from Cenex Supply and Marketing, which sells expertise, financing and farm supplies in the West and Midwest. They call Martin a troublemaker and fear she's fomenting a scare akin to the Alar alarm that nearly ruined Washington's apple industry in 1989. In that case, the CBS television show "60 Minutes" reported that a substance sprayed on Washington apples to preserve them in packing was dangerous to consumers. CBS later admitted it had made some mistakes in the story, and the

Washington apple growers sued the network. But the suit was dismissed, and in the end, Alar was classified by EPA as a carcinogen and banned for all food uses.

"We had a woman starting that one, too, and a lot of people got hurt by it," Bill Weber, an apple and potato farmer, said at one council meeting, bringing nods and laughter. "We don't see a problem," said Greg Richardson, Quincy-based president of the Potato Growers of Washington and a staunch defender of recycling wastes into fertilizer. Richardson wrote Martin a letter telling her to make "a statement of your trust in the appropriate government agencies and their ability to deal with
. . . the waste in fertilizer issue." Martin is standing firm, and a dozen or so Quincy-area farmers are standing at her side. They insist they, their families and their fields have suffered from bad fertilizer.

State environmental, agriculture and health officials have looked at the situation in Quincy. The environmental and agriculture officials, who encourage recycling waste into fertilizer, say that as far as they can tell, there's no danger to crops or people. But some admit they wish they knew more. Kashani wants standards for heavy metals in fertilizer. Absent that, he said, he has to apply a general standard that recycled products cannot "pose a threat to public health or the environment." Regulators in California have been studying the issue for years and still cannot say what constitutes a safe level for lead, cadmium and arsenic in fertilizer. Mayor Martin's husband works for a potato processor, and when she feels under the harshest attack, he tells her she's doing the right thing. "I just have the unfortunate distinction of having stumbled across this question and asking questions
of the regulatory agencies," she said. "I didn't get the answers."

Trouble was brewed in pondHow Martin and her supporters stumbled upon the discovery of the recycling of toxic waste into fertilizer begins at a man-made, concrete pond across the street from Quincy High School. The pond, 36 feet wide, 54 feet long and 5 feet deep, was built in 1986 and used by Cenex to rinse fertilizer from farm equipment. State investigators later found that the company also illegally used the pond to dump pesticides. Cenex closed the pond in 1990. By then, it contained about 38,000 gallons of toxic goo, with heavy metals, suspected carcinogens, even
some radioactive materials. State investigators couldn't determine how all this toxic material ended up there. Cenex memos show how the company got rid of the sludge. John Williams, the Quincy branch manager, wrote his boss to say the "product," as he called it, would cost $170,000 to ship and store at the Arlington, Ore., hazardous-waste site, as required by federal law.

So Cenex decided to save money by spreading it on a rented plot of cornfield and let nature take its course. The land would act as a natural filter for the hazardous wastes. Cenex struck a deal with lessee farmer Larry Schaapman. He was paid more than $10,000 to let Cenex put the material, which the company claimed had fertilizer value, on his 100 acres. It killed the land. The corn crop failed there in 1990, even though Schaapman and Cenex applied extra water to try to wash the toxics through the soil. Hardly anything grew there the next year, either. The land belonged to Dennis DeYoung, whose family had farmed it since the early 1950s before he leased it to Schaapman. Since the land was poisoned, DeYoung couldn't make his payments, and the company that financed him foreclosed on a $100,000 debt. DeYoung also owed Cenex money for fertilizer and seed.

Soon after, Cenex bought the land from the financing company. "They run a farmer out of business, then they get his land," DeYoung said. "Now isn't that something." DeYoung sued Cenex for damages for ruining the soil, lost in summary judgment but won a reversal in the State Court of Appeals earlier this year. He's preparing for a new trial. He also managed to stir up an

investigation by the federal Environmental Protection Agency, which regulate
a plea bargain, Cenex and its manager were given one year of probation for i'
pesticide in the "product" spread on DeYoung's land. The company never h
the heavy metals - enough cadmium, beryllium and chromium to qualify as
got into the rinse pond in town.

That's where Martin and her supporters come in. Farmers began comparin
53-year-old farmer with 200 acres and about 100 cows a few miles east of
purchased the farm in 1956. Witte had a disastrous year in 1991. His red
corn and grain corn all yielded about one-third the normal levels. "You al
you know," Witte said. "You always think you screwed up. But then it wasn't just the
Then I started having all these weird problems with the cows." Six of his cows got sick and died. The
veterinarian found cancer in the three that were tested. When Dennis DeYoung told Witte about his problems, Witte got to wondering about the effects of fertilizer on his fields. Although he hadn't used material from the rinse pond, he had used products from Cenex. Witte still had the rusty, steel fertilizer tank Cenex had delivered and set up on his property in 1991.

Witte reached in the tank and scooped about two pounds of dust, rust and residue from the bottom. He sent the material to Brookside Farms Laboratory in Ohio, which found levels of arsenic,
beryllium, lead, titanium, chromium, copper and mercury. A reporter showed Max Hammond, the
top Cenex scientist in the area, the test results last fall. Hammond, since deceased, said some of the metals might have come from dust or rust in Witte's tank, but he could not explain the beryllium or arsenic. Arsenic, a known carcinogen, is a highly toxic residue from mining and smelting processes. Mayor Martin, who had been closely tracking the rinse-pond controversy, caught wind of Witte's and DeYoung's problems.

Martin, Witte, DeYoung and others began researching fertilizer manufacturing. In their reading, they discovered that, as a result of landfill costs and the stringent environmental laws of the 1970s, a lot of heavy industries were recycling and marketing their hazardous waste as fertilizer. In their
research, they came upon an Oregon lawsuit they think provides a critical insight to Quincy's problems. Aluminum case was studiedNorthwest Alloys, a subsidiary of the Aluminum Company of
America (Alcoa), has a smelter in Addy, an hour's drive north from Spokane. Between 1984 and 1992, the company recycled more than 200,000 tons of hazardous waste from the smelter through
a smaller company that sold it as a fertilizer and road de-icer. Based on industry research that said the material was safe, state officials in Washington, Oregon and Idaho allowed the waste to be sold as "CalMag" and "AlMag" fertilizers and "Road Clear" de-icer. The fertilizer was produced and marketed by L-Bar Products Inc. of Chewelah, near Addy.

With the recycling, Alcoa saved at least $17 million in disposal costs, according to company documents, and many farmers used the products with apparent success. But one Oregon farmer who used it saw his red-clover crop mysteriously wilt. In 1993, he hired James Vomocil, an Oregon State University soils expert, to test his fields and fertilizers. Vomocil said L-Bar's sales flier was
"designed to deceive" and the product was volatile, unpredictable and unsafe. With that ammunition, farmer Wes Behrman of Banks, Ore., won an out-of-court settlement from L-Bar. He
refused to discuss terms of the settlement; he has told other people it was substantial. So

what did that have to do with Quincy? Perhaps nothing. Cenex managers in Quincy and in its regional office say they never bought anything from L-Bar Products and had never even heard of
the company, according to Cenex spokeswoman Lani Jordan.

But a 1994 fax from L-Bar owner Frank Melfi indicates otherwise. It says Cenex had already bought the L-Bar product and was considering buying 30,000 tons that year in "some sort of mutual marketing or venture relationship." Although that deal never happened, Melfi says now that he
definitely sold CalMag to Cenex. Mayor Martin thinks some of it wound up on fields in Quincy, among a variety of other recycled hazardous wastes. And although Cenex denies buying recycled wastes from L- Bar, it has bought material from Bay Zinc to add to custom fertilizer mixes, said Pete
Mutschler of Cenex. But Mutschler said the company didn't realize the Bay Zinc fertilizer contained recycled hazardous waste. Dennis DeYoung began to wonder if fertilizer was to blame
not only for his recent problems, but also for his land turning unproductive in the late 1980s, the reason he decided to lease it to Schaapman in the first place.

At the time, his corn, beans and hay were going bad and he didn't know why. And the more he and others read about what went into recycled fertilizers, the more they began to worry about possible health effects. Martin encouraged Witte and DeYoung to submit hair samples to a Chicago laboratory that tests for heavy metals in human tissues. The lab, Doctor's Data Inc., found high levels
of aluminum, antimony, lead, arsenic and cadmium in hair samples from DeYoung, Witte and Witte's children. Joseph DiGangi, a scientist with Greenpeace in Chicago, reviewed the hair samples. "I thought it was kind of creepy, really - all the people, really headed for a serious health problem, if not now, then later," he said. And it was all perfectly legal.

"It's amazing that something like this could run across the nation and nobody would know about it," DeYoung said. Martin, Witte and DeYoung felt their discovery explained the heavy metals found in Witte's crops. They wondered if the toxic metals in the Cenex pond came from fertilizer residues rinsed from equipment, a theory Cenex vigorously denies. Most importantly, the mayor and farmers knew that while they might never sort out exactly what had happened in their town, they had discovered something other farmers and consumers deserved to know about.
"This recycling might be great in theory, but in fact it's being abused," Martin said.

"There's no enforcement. Nobody is watching the companies. Nobody can tell me what's really happening. Nobody knows." Frustration grew For a man with rough hands and dirty shoes, Tom
Witte writes a good letter. "The state has no mechanism set up to prevent toxic heavy-metals contamination of fertilizers," he wrote then-Gov. Mike Lowry last year. "Fertilizer is only tested for fertility elements. Nobody checks on what is in the inert ingredients, so we have a situation tailor-made for abuse. "People in industry think that the best way to dispose of waste is to sell it for fertilizer and let unsuspecting farmers spread it on their land."

Agriculture Director Jim Jesernig wrote back, agreeing there were problems and promising to look into it further. The departments of agriculture, ecology and health have set up a staff group that plans to issue a report later this year saying the practice, which they have encouraged for years, is safe. State officials say they have tested a sampling of 27 potatoes and that heavy-metal readings were well within safe limits. Meanwhile, Mayor Martin and Witte's sister, Nancy, a

nurse, went to EPA Administrator Carol Browner's Children's Health Conference in Washington, D.C., in February. Nancy Witte prodded a nervous Martin to go to the microphone and ask a question of Browner. Martin asked whether the EPA knew about companies making toxic wastes into fertilizer. Browner said she didn't know anything about it but she'd look into it. Later, an aide to Browner contacted the mayor, explained the benefits of waste recycling and assured her there would be further study.

Frustrated with the lack of action by public officials, Martin contacted The Times, asking the newspaper to develop this information. Potential for danger unclearSo what to make of Mayor Martin and her crusaders? Are they, as Richardson of the Potato Growers of Washington insists, unnecessarily "opening up an ugly can of worms"? All that's clear is that the potential for danger is unclear. Some scientists and public officials say the benefits of recycling waste outweigh the possible
risks. "The farmer is coming out a little ahead," said soils specialist Charlie Mitchell of Alabama's Auburn University. "The person spreading it is getting his profit. The company is using its waste instead of dumping it. So we're helping the environment. We're creating jobs. If it's done right, it can really be a win-win situation." But Ken Cook, a soils scientist who heads the nonprofit Environmental Working Group, said no one yet knows what constitutes "doing it right."

Mayor Martin and friends are raising good questions, Cook says. "Let's put it this way: We're well into the use of these materials before these questions are even asked, and that doesn't seem to me to be a good sign that we've been very rigorous in our science on this." Meanwhile, Quincy farmers such as Witte, DeYoung and Duke Giraud want some action. Giraud lost his family's onion business because of poor yields, and he suffers from respiratory problems. He figures he unknowingly spread recycled-waste fertilizer on his fields. It might be too late for him, he says, but he wants government agencies to look out for the welfare of other farmers. "They have to start testing fertilizer for what they don't say is in there," Giraud says, "because they have no problem letting them add who-knows-what." Let us know what you think. E-mail Duff Wilson at dwil-new@seatimes.com What's known, and not known, about toxics, plants and soilResources on the World Wide WebHeavy Metals in Fertilizers

BACK

The Practice of Using Hazardous Waste as Fertilizer

It has become common practice for the wastes from certain industries, such as steel mills, smelters, and pulp mills, to be sold as fertilizer. Some industrial wastes contain chemicals which are beneficial to plants, such as zinc and liming agents.However, these materials may also contain high levels of heavy metals, dioxins, or other toxic chemicals that have no nutritive value to plants and are known to be harmful to human health. This fact was not widely known or even addressed publicly until recently.

Fertilizers in the United States are regulated only for nutrient content. Industrial wastes are no longer subject to federal hazardous waste regulations including those governing disposal if they are marketed as fertilizer. Therefore there is a strong economic incentive for companies producing hazardous waste to avoid disposal costs and potential liability by repackaging it as fertilizer.

Farmers in Quincy, Washington and the former Mayor Patty Martin began asking questions about this practice that led to an investigation by the Seattle Times and legislative action by the Washington State legislature. Although the practice is widespread across the country, Washington State is the first to pass legislation that attempts to address this issue.

Case Studies: Quincy, Washington

Several farmers in the area near Quincy in Central Washington believe their land has been contaminated with toxic fertilizer. One farmer, Dennis DeYoung, lost a 100-acre tract of land that had been in his family for forty years, when a crop was ruined and the bank foreclosed on his land. DeYoung had leased the land to a farmer, who accepted $10,000 in payment to apply a mixture of toxic chemicals which the local fertilizer company Cenex claimed had fertilizer value. The chemicals came from a pond operated by the company used to rinse fertilizer from farm equipment. The company also illegally dumped pesticides in the pond. When it was closed in 1990, it contained 38,000 gallons of toxic chemicals including heavy metals, suspected carcinogens, and some radioactive materials, including cadmium, beryllium and chromium at levels high enough to be classified as a Superfund site. The company would have had to pay $170,000 to ship and store the material as hazardous waste at a site in Oregon. Instead, it was applied to DeYoung's land, and the land was supposed to act as a "natural filter" for the hazardous wastes. Instead, the corn crop that year died. As DeYoung was unable to make payments, the company that financed the land foreclosed on the debt, and the land was subsequently bought by Cenex. (Wilson, 1997)

Witte, another farmer in the area, had a supply of fertilizer from the same company tested after his crops failed and three of his cows died of cancer in one year. The fertilizer was found to have levels of arsenic, beryllium, lead, titanium, chromium, copper, and mercury. Witte and his children, and DeYoung had hair samples tested for heavy metals, and high levels of aluminum, antimony, lead, arsenic, and cadmium were found in all of them. (Wilson, 1997)

Across the Country

Similar cases have been documented by The Seattle Times in other states including California, Georgia, South Carolina, and Oklahoma. It has been reported that some farmers are reluctant to speak out about contamination problems for fear that they wouldn't be able to sell their produce or land. Many farmers have land mortgages from the same companies that sell them agricultural chemicals. (Wilson, 1997)

The extent of this practice was studied by the Environmental Working Group in their report "Factory Farming: Toxic Waste and Fertilizer in the United States, 1990-1995." The study analyzed data from the federal

government's Toxic Release Inventory (TRI), and found that between 1990 and 1995, a total of 271 million pounds of hazardous waste were sent to 454 companies identified as farms and fertilizer companies. The major sources of waste were steel mills, foundries, and electronic component manufacturers, with wastes from the steel industry comprising 30% of the total volume.(EWG, 1998)
The TRI data does not indicate how much of the wastes were made into fertilizer at the receiving facilities, or how toxic the finished products were. It also does not provide information on the use of toxic wastes that were sent to farms or individuals for land disposal, "other" recycling, or land application, or whether food crops were grown at those locations. On the other hand, most toxic waste produced in the U.S. is not tracked by the TRI, including many wastes contaminated with dioxin, and so this data set may underestimate the extent of this practice.(EWG, 1998)

(EWG, 1998)

Overview - Contaminants and Health - Soils and Toxics - Regulations - Conclusions - Links - References

Recycling hazardous waste into fertilizer causes alarm

Agricultural threat: There are no regulations regarding the amount of lead, cadmium or other toxics put into fertilizers.

The Associated Press

SEATTLE - Toxic heavy metals, chemicals and radioactive wastes are being recycled as fertilizer and spread over farmers' fields nationwide - and federal law doesn't require that they be listed as ingredients, a Seattle newspaper reported.

The issue came to light in the central Washington town of Quincy, population 4,000, when Mayor Patty Martin led an investigation by local farmers concerned about poor yields and sickly cattle.

"It's really unbelievable what's happening, but it's true," Martin said.

Until now, the state Department of Agriculture sampled fertilizers only to see if they contained advertised levels of beneficial substances.

But the state is currently testing a cross-section of fertilizer products to see if they threaten crops, livestock or people.

"The key question is what toxics are, as it were, along for the ride in fertilizers," said Tom Fitzsimmons, director of the state Department of Ecology.

Use of industrial waste as a fertilizer ingredient is a growing national phenomenon.

In Gore, Okla., a uranium processing plant gets rid of low-level radioactive waste by licensing it as a liquid fertilizer and spraying it over 9,000 acres of grazing land.

In Camas, lead-laced waste from a pulp mill is hauled to farms and spread over crops destined for livestock feed.

In Moxee City, dark powder from two Oregon steel mills is poured from rail cars into silos at Bay Zinc Co. under a federal hazardous waste storage permit. Then it is emptied from the silos for use as fertilizer. The newspaper called the powder a toxic by-product of steelmaking but did not identify it.

"When it goes into our silo, it's a hazardous waste," said Bay Zinc's president, Dick Camp. "When it comes out of the silo, it's no longer regulated. The exact same material."

Federal and state governments encourage the recycling, which saves money for industry and conserves space in hazardous-waste landfills.

The substances found in recycled fertilizers include cadmium, lead, arsenic, radioactive materials and dioxins, the newspaper reported. The wastes come from incineration of medical and

municipal wastes, and from heavy industries including mining, smelting, cement kilns and wood products.

Mixed and handled correctly, some industrial wastes can help crops grow, but beneficial materials such as nitrogen and magnesium often are accompanied by dangerous heavy metals such as cadmium and lead.

From the Olympian, July 7, 1997
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Contaminants and Associated Health Effects

The exact effects on human health and the environment from the use of hazardous waste as fertilizer are not known. However, some of the chemical components of these materials such as heavy metals and dioxins have been extensively studied and have been been shown to cause serious harmful effects. Plants can take up heavy metals from the soil and concentrate it through bioaccumulation. For some metals, food intake is the main source of exposure.

Testing Results for Metals and Dioxins in Fertilizer

In 1997, the Washington State Department of Ecology conducted a screening study of thirty five fertilizers, including six that were made from industrial by-products. Ecology tested for the nine metals for which biosolid standards have been developed, plus an additional 15 metals, plus dioxin, a by-product of industrial processes.(Ecology, 1997)

The study found that levels of heavy metals varied considerably. For lead, values ranged from 11 parts per million (ppm) to 11,300 ppm. Cadmium levels ranged from .6 to 275 ppm. Of the five fertilizers made from industrial waste tested for dioxins, all samples were found to contain dioxin. Results were expressed in toxic equivalents, or TEQ's. This is a standard way to express concentrations of dioxins in the environment. This value is derived from summing toxicity values of the 17 forms of dioxin considered to be toxic. TEQ concentrations of the samples ranged from .59 parts per trillion (ppt) to 815 ppt. (Ecology, 1997) In comparison, the Washington State standard used to clean up dioxin at hazardous waste sites is 6.67 ppt. (WTC, 1998)

Health Effects

Below are some health effects associated with three of the most dangerous contaminants found in fertilizer made from hazardous waste; lead, cadmium, and dioxins.

Table 1. **Health Effects Associated with Some Contaminants**

Contaminant	Health Effects	Exposure	Env. Fate
Lead	Permanent neurological damage, endocrine system disruption	-mainly from fruits and grains -deposition from air to plants, livestock eating soil -no known safe level	persistent
Cadmium	cancer, kidney disease, neurological disfunction, fertility problems, immune system changes, birth defects	-mainly through food	persistent, bioaccumulative
Dioxins	cancer, endocrine disruption, immune system damage; negative effects seen at levels as low as ppt	-mainly through meat and dairy consumption	persistent, bioaccumulative

Sources: Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Environmental Protection Agency

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Colorado covers CWD disease for 12 ye...

Author	Message
Dave Member Username: Dave Post Number: 274 Registered: 1-2002	Posted on Friday, January 17, 2003 - 8:03 am: Jeff Van Steeg, division of Wildlife's top game manager said it was a bad call covering up for 12 years the fact they new they had moved infected elk. Pretty easy just to blame it on the elk farmers. I wander if WI will ever admit to bringing elk to WI was a bad move knowing they came from a herd that has tested positive for TB. http://www.trib.com/AP/wire_detail.php?wire_num=60771
Diane Morison Guest	Posted on Friday, January 17, 2003 - 12:10 pm: CWD-exposed elk used in 1990 study Wildlife officials call W. Slope move a mistake By Theo Stein, Denver Post, January 17, 2003 http://www.denverpost.com/Stories/0,1413,36%257E11799%257E1116921%257E,00.html The Colorado Division of Wildlife knowingly used a herd of captive elk exposed to chronic wasting disease in a grazing study on the Western Slope in January 1990, possibly introducing the disease to the elk-rich area. "It was a bad call," said Jeff Ver Steeg, the division's top game manager. "I can't deny it." About 150 wild elk were allowed to graze in the same pens near Maybell after the research herd was removed and may have picked up the abnormal protein that causes the disease from the feces and urine left by the captive elk. While the Division of Wildlife has expressed concern before that its animals might have helped spread CWD, this is the first time the agency has acknowledged it knowingly moved elk exposed to CWD deep into an area where the disease was not known to already exist. Studies that could help determine the source of CWD on the Western Slope are incomplete, and officials say what data that do exist are so new and so spotty they may not provide all the answers. So far, it appears that less than 1 percent of deer and elk in the area are infected, compared with as much as 15 to 20 percent in hotspots in northeastern Colorado. But as wildlife officials grapple with CWD's appearance in northwestern Colorado, officials now admit the decision to continue the grazing study over the objections of some biologists was an error. At the time, biologists wanted to see whether elk grazing on winter

range depleted forage that ranchers wanted for fattening cattle in spring.

"I think in hindsight a lot of good people probably did some dumb things, myself included," said Bruce Gill, a retired wildlife manager who oversaw research efforts and remembers the debate over the project. "Had we known CWD would explode into such a potentially volatile ecologic and economic issue, we wouldn't have done it."

Elk ranchers, who have been blamed for exporting the disease from its stronghold on the Colorado and Wyoming plains to seven states and two Canadian provinces, say the agency's belated disclosure smacks of a coverup.

"It's pure negligence," said Jerry Perkins, a Delta banker and rancher who is now demanding a legislative inquiry. "If I'd have moved animals I knew to be infected around like that, I'd be in jail."

Grand Junction veterinarian and sportsman Dick Steele said he faults the agency for not disclosing information about CWD-exposed research animals before October, when information was posted on the Division of Wildlife website.

"This went way beyond poor judgment," he said. "My main concern is that this has been hidden for the last 12 years. It would have been real important to our decision-making process on how to deal with CWD."

While the Maybell information is new, Perkins and other ranchers have long suspected Division of Wildlife research facilities near Meeker and Kremmling, which temporarily housed mule deer kept in heavily infected pens at the Fort Collins facility, have leaked CWD to the wild.

Fear of an outbreak led the agency to sample 450 deer around the Meeker and Kremmling facilities. None tested positive, but the sample size was only large enough to detect cases if the infection rate was greater than 1 percent.

This fall, tests on 23,000 deer and elk submitted by hunters statewide have revealed 48 CWD cases north of Interstate 70 and west of the Continental Divide.

Biologists believe the infection rate in that area, which includes the Maybell, Meeker and Kremmling sites, is still well below 1 percent. But CWD has never been contained in a wild population, so experts fear the problem will grow worse.

The Division of Wildlife says it will be months before a statistical analysis of the fall's sampling results can be completed, an exercise that may shed light on the disease's origin on the Western Slope.

"We're just not going to speculate at this point," said Ver Steeg of the possible Maybell connection. "This is one possibility, but certainly not the only possibility."

Some biologists think a defunct elk ranch near Pagoda, which had dozens of unexplained deaths in the mid-'90s, is another, a suggestion Perkins rejects.

"It may be inconclusive to them," said Perkins. "It isn't inconclusive to us."

Donald
Guest

Posted on Friday, January 17, 2003 - 5:53 pm:

One CWD positive Elk was brought into Arkansas via hunter also. AG&F confirmed this. Arkansas also brought in some elk from CO. they say they were Rocky Mountain Elk but not from the Eco Valley Ranch.

Donald
Guest

Posted on Friday, January 17, 2003 - 5:55 pm:

One CWD positive Elk was brought into Arkansas via hunter also. AG&F confirmed this. Arkansas also brought in some elk from CO. they say

Four thousand tons of ash from a U.S. garbage incinerator are sitting in a beach town in Haiti right now. A ship carrying this ash from the city of Philadelphia dumped it there 10 years ago, claiming it was fertilizer. The heavy metals and other toxics in the ash are blowing in the tropical wind and being carried into the sea by heavy rains. Cadmium and lead, two metals present in the ash, contribute to neurological damage, lung and bone disorders, birth defects and other health problems. Samples of adjacent soil show the toxics have migrated into the environment. In ten years, not even a fence or warning sign has been erected to protect the community.

Haitian and U.S. environmentalists have been working for years to persuade the Philadelphia and U.S. authorities to repatriate the ash without success. But just when Haitians and U.S. activists where about to give up, a solution is in sight. This web site is devoted to PROJECT RETURN TO SENDER, and a fighting chance that the toxic waste dumped in Haiti will finally be coming home. But we need your help to make it happen.

An Analysis of Risks Associated with the Disposal of Deer from Wisconsin in Municipal Solid Waste Landfills

1. Background

The purpose of this document is to evaluate risks associated with the landfilling of CWD-infected deer carcasses. It uses the current scientific information available on this subject and, as such, should be considered an interim document that will be updated as new information is available.

1.1 The Challenge

In February 2002 the first cases of chronic wasting disease (CWD) in free-ranging white-tailed deer east of the Mississippi River were reported in southwest Wisconsin. Further surveillance revealed a 3% rate of infection in an 11-mile radius around the initial cases. Based on these findings and the input of wildlife disease control and CWD experts, a disease eradication program was developed. This program calls for the harvest of all of the deer within a 360 square mile area and for population reductions in the surrounding areas.

The zone targeted for complete depopulation has been designated the *eradication zone* (EZ) and the surrounding area has been termed the *management zone*. It is estimated that, as of fall of 2002, there are approximately 25,000 deer in the EZ alone. Citizen-hunters are expected to be the primary means of removing deer from the EZ. It is expected that the majority of these deer will not be used as a source of venison. Therefore, the carcasses will need to be disposed of in a manner that does not jeopardize animal or human health or environmental quality.

Any disposal method must also have the following attributes: the capacity to handle a large number of carcasses; the ability to conform to local, state and national laws and regulations; and to be in place by October 2002. A final consideration is the cost of disposal. Although the latter is not the primary consideration, it is likely that disposal costs will be one of the largest expenses of Wisconsin's CWD control program. The four primary options currently under consideration for the disposal of deer are landfilling, rendering, incineration and chemical digestion.

Deer carcasses and tissues are often sent to municipal solid waste landfills. This material is incorporated in with other waste at the landfill. Landfills generate a certain amount of liquid, termed *leachate*, which is collected and processed. This leachate results primarily from precipitation falling on the landfill surface. Composite landfill liners prevent leachate from entering groundwater. The leachate is collected at the base of the waste just above the liner. In most instances the leachate is transferred to a wastewater treatment plant (WWTP) for treatment. Less commonly, some landfills may recirculate a portion of the leachate. At the WWTP, the leachate is processed along other wastewater. Solids are separated from the water portion. This material, termed "sludge" or biosolids, is commonly applied on farm fields or landfilled.

Based on the above, the primary pathway of potential risk identified for the CWD prion following landfill disposal of infected deer can be described as:

carcass ▶ landfill ▶ leachate ▶ wastewater treatment plant ▶ sludge ▶

farm field► ingestion by humans or deer.

1.2 The Disease

CWD is a member of the transmissible spongiform encephalopathies (TSEs) a group of diseases that includes scrapie of sheep, bovine spongiform encephalopathy (BSE) of cattle and Creutzfeldt-Jakob Disease of humans. BSE is the only animal TSE for which there is experimental and epidemiological evidence of transmission to humans (Bruce *et al.* 1997, Hill *et al.* 1997 and Scott *et al.* 1999). All of the diseases in this group are characterized by a prolonged incubation, insidious onset of neurological signs, typically slow progression and eventual death. As a group, the TSEs are infectious, but not highly contagious. The specific transmission routes (i.e. portal(s) of agent exit and entry) of CWD between infected and susceptible animals have not been established. There is evidence that CWD can be transmitted by direct and indirect means; that is by animal-to-animal contact or by animal contact with contaminated items or the environment.

1.3 Biochemical and Physical Properties of the TSEs

Biochemically, the TSEs are characterized by a resistant form of a normal protein that is found in all mammalian and avian species examined to date. This protein is termed *prion protein* (PrP). The abnormal form, termed *PrP-res*, is associated with TSE infectivity and pathogenicity. The "res" refers to the fact that the abnormal prion protein is partially resistant to proteinase K digestion. Unlike the normal host prion protein, PrP-res forms ordered oligomeric structures; which are units composed of more than one protein chain. PrP-res has both hydrophilic and hydrophobic regions (Meyer *et al.* 1986). The hydrophobic region will be an important determinant of the behavior of the CWD agent in the landfill environment. Other distinctive properties of PrP-res include resistance to many of the commonly used disinfectants and inactivation procedures that are typically used to destroy infectious agents. Finally, there are multiple strains of TSE agents that have been identified, even within a particular disease group. Research has shown that some strains are more resistant to inactivation than others. With respect to CWD, it is unknown whether there are multiple strains and what relative degree of resistance to inactivation CWD has with respect to some of the better characterized TSE agents such as scrapie and BSE.

2. Behavior of the Prion Protein (PrP-res) in the Environment

2.1 Soil and Solid Waste

Due to the hydrophobic regions of the CWD PrP-res molecule, infectious prions in the environment can be expected to adsorb to organic material and soil. Initially the infectious agent is likely to adhere to the protein and carbohydrate components of the animal carcass. As the carcass decomposes, the undegraded PrP-res will adhere to adjacent soil or waste material in a landfill. The ability of scrapie prions to bind to metals and plastics has been reported (Flechsig et al, 2001; Weissman et al., 2002). All municipal solid waste landfills in Wisconsin employ a thick plastic membrane (generally polyethylene) as a component of the liner. This liner acts as a protective barrier and should prevent the movement of 'free' prions to subsurface soils or to groundwater.

In the only experiment to examine the fate of PrP-res in an outdoor environment, Brown and Gajdusek (1991) buried perforated petri dishes containing hamster scrapie in a residential garden for

three years. They found that approximately 1% of the original infectivity in the original location survived this term. Examining surrounding soil layers, no infectivity was found above the original location, a small amount of infectivity was found in the 4 cm soil layer that was directly beneath the perforated dish containing the original inoculum and no infectivity was found at 4-8 cm below the dish. The authors conclude that the hamster scrapie agent used in this experiment can persist in contaminated soil for three years under natural environmental conditions, but that there is little leaching to surrounding soil layers. To date there has been no further work that specifically examines the fate of TSE agents in the soil/solid waste environment.

Land application of municipal sludge that potentially contains CWD PrP-res may result in the presence of CWD PrP-res in surface soils. The application rates of municipal sludge are dependent on the chemical characteristics of the sludge and therefore will vary. The mechanism and time course of PrP-res degradation in soil/solid waste environment is unknown. Normal biodegredation processes are expected to inactivate the CWD prion over time.

2.2 Water

The hydrophobic nature of PrP-res (Bennett 1992) indicates that leaching of the CWD agent into an aqueous environment is unlikely to occur in the landfill or in soil. This assumption is echoed in a 2000 BSE risk assessment produced for the British Ministry of Agriculture, Fisheries and Food (now Department for Environment, Food & Rural Affairs [DEFRA]). In section 3.1 titled "Fundamental Assumptions" it states that "BSE agent is stuck to particulate matter and, hence, is removed with the particulate matter from the effluent." Gale *et al.* (1998) in examining the risk from BSE in the aquatic environment state, "With the possible exception of flows in the vicinity of extraction wells, the rate of flow through landfilled wastes is generally slow and non-turbulent, with the result that particulate material is unlikely to be taken up in suspension."

Should any PrP-res exit the landfill as part of the leachate, it will, due to its hydrophobic nature, be attached to particulates (colloids) suspended in the leachate. Once that leachate reaches the wastewater treatment plant the suspended solids will be separated from the effluent. Those suspended solids will then be termed "sludge" or biosolids. Again, due to its hydrophobic nature, the PrP-res is expected to selectively partition with the solids into the sludge portion, and is not expected to be present in wastewater discharged to surface water. Gale and Stanfield (2001) discuss this expectation in their risk assessment for BSE in sewage sludge.

2.3 Air

Air transport is not considered in this document because there is currently no evidence that PrP-res can be released into the air or volatilized in any way under natural conditions. In addition, there is no evidence of airborne transmission between animals or people.

3. Human vs. Animal Exposure to the CWD Agent

To date, no human illness has been associated with exposure to the CWD agent. However, systematic surveillance has only recently begun. Given that humans have likely been exposed to the CWD agent for decades from animals, in laboratories and from the environment, this is a significant observation. Surveillance of prion-related diseases in humans is in its infancy. The primary routes of exposure in the future are likely to be through hunting and the consumption of CWD-contaminated

venison and elk. To date, test-tube experiments in which normal human prion protein is exposed to PrP-res from white-tailed deer have shown a limited degree of infectivity, but at a less efficient rate than that for BSE or scrapie (Raymond et al., 2000).

In contrast to interspecies transmission of CWD from deer to humans, there is good evidence that deer and elk can contract CWD by animal-to-animal contact as well as by contact of a susceptible animal with a contaminated environment.

Domestic cattle have failed to develop disease when housed with CWD-infected deer (Williams & Miller 2002). Under experimental conditions, however, 3 of 13 cattle inoculated (~ 5 years ago) intracerebrally with CWD did succumb to a TSE illness (Hamir et al., 2001). The remaining cattle are still alive and will remain under observation for another 5 years. Animals orally inoculated have, to date, not succumbed to the disease (Williams, 2002). Based on the above observations, the following pathway is not considered further in this document:

deer ▶ landfill ▶ leachate ▶ wastewater treatment plant ▶ sludge ▶ farm field ▶ animal fodder (surface contamination) ▶ domestic livestock ▶ commercial meat.

4. Impact Assessment

4.1 Minimum Level of Exposure Known to Cause Disease

As described above, it is likely that over the past several decades, thousands of hunters, taxidermists, meat processors, and research staff have ingested the CWD prion, as well as been exposed via eye splashes and through cuts and wounds. Exposure from these routes is likely to have been significantly greater than any that would be expected to result from contact with leachate from a well-managed landfill.

The issue of how much infected material an individual (human or animal) must consume or be exposed to in order to become infected with CWD or any other TSE is not known. Neither is it known if repeated small doses can result in infection. In an experimental setting, low-dose inoculation studies have revealed a decreased probability of infection and prolonged incubation periods. In some animal experiments, the incubation period extended beyond the natural life span of the animal; that is, at the time of death due to "natural causes", the animal was infected with the TSE agent, but was not symptomatic (Dickinson, 1977; Thackray et al., 2002).

4.2 Transmission of TSEs to Humans and Among Animals of the Same Species

As stated in section 2., the only animal TSE that is known to have been transmitted to humans is BSE. Transmission of TSEs from one animal to another is likely to depend on a number of factors. These include the specific TSE, the strain of the TSE, the dose, the route of exposure, the human PrP genotype, and likely other unidentified factors.

The dose for any TSE is typically expressed as a "infectious/lethal dose 50" or an "I/LD_{50} per gram of tissue. It represents the dose of material at which 50% of the recipients become infected and will die. An I/LD_{50} is always species and route specific. The I/LD_{50} is determined by serial dilutions of the original material and subsequent inoculations into groups of animals to determine the endpoint at

which 50% of the animals succumb or are diagnosed as infected (The term "LD_{50}" is often replaced by "ID_{50}" or "infectious dose 50" to indicate that experimental animals are not allowed to progress fully through clinical disease to death). For the TSEs, a lower dose can decrease the likelihood that an exposed animal will become infected.

The route of exposure is also an important factor. The intracerebral route of inoculation is the most efficient. However, it is not a natural route of exposure. The following additional routes of infection are listed in descending order of efficiency (generally): intravenous, intraperitoneal and oral. The oral infectious dose of CWD has not been determined for deer or elk (E. Williams, personal communication, 2002).

4.3 Movement of Prions to Landfill Leachate

While the assumptions in this analysis are based on limited data, they serve to provide an approximation of the range of conditions likely to be encountered in the environment under the scenario described in this document.

4.3.1 Permeability

Permeability is defined as the time needed for liquids to percolate through the waste mass at a landfill. There are limited data available regarding the saturated hydraulic conductivity of municipal waste. The EPA Hydrologic Evaluation of Landfill Performance (HELP) model for predicting the movement of liquids through landfill caps and liners uses a default value of 1×10^{-3} cm/sec for the saturated hydraulic conductivity of municipal waste. This value is based on work by Oweis et al (1990). More recent data (Shaw and Carey, 1996, Bleiker et al,1993;Townsend et al,1995) indicates a broader range of permeabilities from 10^{-3} to 10^{-6} cm/sec. Permeability, however, can vary based on waste composition, age (degree of decomposition) and depth of fill.

4.3.2 Distance to the Leachate Collection System

Typical municipal waste landfills in Wisconsin range in final height from 100 to 300 feet above the leachate collection system. The landfills are typically constructed in a series of phases over time, so that there are disposal areas available in the upper portions of the previous phase, while filling is occurring at the base of a new phase. DNR recommended in a June 6, 2002 letter to landfill operators that the deer carcass burial area should be "strategically sited high in the landfill such that any liquids will have to pass through many feet of waste material before reaching the leachate collection system"(WDNR, 2002).

4.3.3 Summary

Considering these factors in tandem with the observations in section 2.1 about the hydrophobicity of prions and their tendency to degrade in soil, it is expected that if prions were to move into landfill leachate, their movement would be slow enough that their concentration would be significantly reduced by degradation and retention in the remaining waste mass.

4.4 Ingestion of potentially CWD-contaminated soil by humans and deer after sludge application

As described in section 2 of this document, it is expected that any prions present in leachate will adhere to sludge during the wastewater treatment process. Furthermore, the incorporation of sludge into the 9-inch plow layer, which is standard for land application practices, would provide significant dilution within the soil. This combination of concentration reduction factors at the landfill, the sewerage treatment plant, and in the soil as well as the natural degradation processes is expected to greatly reduce the potential for infectious CWD prions to be present in sludge-amended soil.

5. Discussion

A quantitative or semi-quantitative assessment of the risk is not possible because the amount of infectivity present in a carcass is unknown. In addition, the dose needed to infect a human or deer is also unknown. Nonetheless, existing information suggests that landfilling large numbers of deer from an area with a low incidence of CWD is unlikely to pose a significant risk to humans or to wildlife.

This document provides support for the following conclusions:

1. The disease specific agent is hydrophobic and is expected to adhere to organic materials present in a landfill.
2. It is likely to take the CWD agent several months to move through a landfill. During that time the agent will be subject to biodegredation and is likely to lose a significant amount of its infectivity. Based on the findings of Brown and Gajdusek (1991), up to 98% loss of infectivity can be anticipated within a 3-yr period.
3. Any infectivity that exits the landfill will be captured in the effluent and transferred to a wastewater treatment plant or recirculated in the landfill.
4. CWD prions present in wastewater are expected to partition with the sludge fraction.
5. Land-applied sludge will be greatly diluted by surface soils and incorporated with soil at a depth of 9 inches.

5.1 Likelihood of Human Exposure

Two factors strongly influence human risk:

1. The presence of a species barrier
2. The route and dose of the exposure

The precise nature of any species barrier for CWD transmission between white-tailed deer and humans has, as yet, not been described. Given the fact that controlled experiments cannot be conducted in human beings, the existence of a species barrier cannot be directly tested. However, there is limited experimental evidence of a species barrier (Raymond et al. 2000). Further supporting the notion that such a species barrier exists is the observation that humans have been handling and consuming tissues from infected deer for decades with no evidence to date of any correlation with any human illness. The U. S. Centers for Disease Control (CDC) has investigated Creutzfeldt-Jakob disease (CJD) among three individuals thirty years of age or younger who had some association with hunting or consumption of venison (not related to the ongoing CDC investigation in Wisconsin) and has concluded that there was no evidence for a causal link with the consumption of venison (Belay et al., 2001).

DNR'S own Paper

The route of exposure to TSEs is also an important determinant of the efficiency of transmission. Oral (i.e. ingestion) exposure is among the least efficient means of transmitting any TSEs. In many circumstances, TSEs that can be transmitted by the artificial route of intracranial (IC) inoculation directly into the brain cannot be transmitted by the oral route. Under experimental conditions when the dose can be controlled, it generally requires a far greater dose (typically 1,000- to 100,000-fold more) to transmit a given TSE at the same rate by the oral route than by the IC route.

The collection of leachate from a large landfill, the co-mingling of the solids from the leachate with other solids from the sewerage system and its mixing with 9 inches of topsoil provides an extremely large dilution factor. In addition, ~~any prions that enter the environment will degrade with time.~~

In summary, it is reasonable to conclude that while absolute numbers relating to human health risk cannot be generated, the available knowledge about CWD and other TSEs suggests that landfilling of CWD-infected deer does not pose a significant risk to human health.

5.2 Likelihood of Transmission to Deer

The major factors that influence the risk to deer from the landfilling of a population of deer some of which are infected with CWD are:

1. The absence of a species barrier
2. The route of exposure
3. The concentration reduction factors inherent in sludge production and application
4. The consumption of soil by deer.

For any TSE that is transmitted within the same species there is assumed to be no species barrier. The consequence of this is that deer are the most susceptible species to any exposure to viable CWD agent that enters the environment. ~~For deer there may, however, be genetic influences on susceptibility and incubation period.~~ For CWD this information is not known. Therefore, the approach in this document has been to assume that all white-tailed deer are equally susceptible to CWD infection.

The primary route of exposure to the CWD prion for deer from sludge amended soil is by ingestion. Typically for the TSEs the oral route is among the least efficient means of transmission. However, the oral infectious dose of CWD for deer has not been determined.

The collection of leachate from a large landfill, the co-mingling of the solids from the leachate with all the other solids from the sewerage system and then its mixing with 9 inches of topsoil provides an extremely large concentration reduction factor. Should any viable CWD prions make it out into the environment it is likely that they ~~will degrade with time~~ and will be diluted due to the mixing to a depth of 9 inches.

In conclusion, it is deemed likely that the risk of spreading CWD among Wisconsin's deer population by landfill disposal of infected carcasses is quite small.

6. References:

Belay ED, Gambetti P, Schonberger LB, Parchi P, Lyon DR, Capellari S, McQuiston

- Disabled Hunters
- Habitat - ie. N. WI Dense Cover vs Central & S. WI Fencelines & Patches of woods

- DNR: Sept. 2002 To: Sanitary Land fills & Public Sewage Treatment Operations.

Darrell Bissell

Subject: Disposal & Landfilling of Deer Carcasses

DNR says oral transmission is least apt. to spread CWD

CWD

Landowner CWD Newsletter

Premier Issue

Volume 1, Issue 1
April 2003

Introduction

Wisconsin state officials have saturated the media with comments in defense of their CWD policy. Their position has been repeated widely and often, and has been backed up forcefully and with massive resources. But that does not make it true. It also does not make it popular with the people most affected, landowners in or near the DNR's total kill zone.

We see things differently than these state officials, and would like to offer our perspective on the matter. We do not think that they have made a good case for taking such drastic and expensive action. And we know the disrespectful and abusive way they have treated citizens/landowners is wrong.

Here you can read about the views of ordinary citizens/landowners who are opposed to Wisconsin CWD policy. Our goal is to give voice to the people, and to send a message to those who work for us in state government. After looking over this newsletter, you be the judge.

We invite your questions, comments and suggestions.

Group Activities

Citizens and Landowners for a Rational Response (CALFARR) was formed in June 2002 by a group of Iowa County property owners who were baffled by the DNR saying it had overwhelming landowner support for its CWD policy. The group has continued to meet every week or so to discuss opposing views to those held by state officials.

Several actions have followed from these meetings. First, a petition was circulated to see which landowners were opposed to CWD policy. The result of this poll is the dramatically revealing "red petition map" that is reproduced on page 4. Second, a lawyer was retained to explore landowners' options in receiving possible damages with regard to the state causing harm and loss to individual landowners. Third, the group created documents, made proposals and attended meetings to get its views out. Personal meetings were held with DNR secretaries Bazzell and Hassett. And last, requests were made under Wisconsin "Open Records" Law to get access to CWD data, methods, and procedures that state officials were otherwise not willing to share.

CWD Facts

- Chronic Wasting Disease belongs to a family of diseases that are characterized by a distorted (twisted/folded) form of a normal protein called a prion.
- It is not known whether prions are a cause or a symptom of CWD.
- The geographical origin of CWD is unknown, but it was first detected in Colorado deer/elk research pens in 1967.
- It is not known how CWD got to Wisconsin or if it has been here all along.
- The agent/factor that leads to CWD is not known.
- There is no evidence that CWD can spread orally between deer through normal feeding, licking or grooming behavior.
- There is no evidence that CWD is a health risk to humans or livestock.
- CWD is slow developing. Current tests cannot detect the disease before the deer/elk is 18 months old.
- CWD is not highly infectious, since there have been only 600 known cases since it was discovered 40 years ago.
- Some deer have lived for 7 years in pens where other animals were infected with CWD.
- In no place on earth has CWD in wild deer ever been eliminated from the population.
- We do not know how many deer that test positive for CWD would eventually die from the disease if left alone.

The scariest thing you can hear from scientists is: "We don't know."

What Experts Say

The number of case submissions [of CWD] has increased since the first case was discovered [in the wild] in 1981. We believe this increased detection rate is mostly due to greater efforts to locate affected animals.

- Journal of Wildlife Diseases, 1997

The duration of CWD occurrence in free-ranging [deer/elk] remains as [puzzling] as its geographic origin. CWD may have been present in free-ranging deer in both Colorado and Wyoming since the early 1960's, if not earlier.

- Journal of Wildlife Diseases, 2000

The biological mechanisms underlying CWD transmission are poorly understood, and as a result model mechanisms [and predictions] are at best a collection of educated guesses.

-Journal of Wildlife Management, 2001

Selective culling may offer the greatest promise of reducing CWD prevalence, particularly when infected populations are detected early in the course of an epidemic and tested aggressively for several decades.

- Journal of Wildlife Management, 2001

Although most captive deer residing in ... research facilities [holding some infected animals] eventually contract CWD, individuals occasionally survive a lifetime. Genetic resistance ... has been demonstrated in free-ranging and farmed elk and is being investigated in deer.

- Journal of Wildlife Management, 2002

The apparent persistence of [CWD] in contaminated environments may represent a significant obstacle to eradication of CWD from either farmed or free-ranging [deer/elk] populations. Perhaps most important, impacts of CWD on population dynamics of deer and elk are presently unknown.

- Journal of Wildlife Management, 2002

What THEY SAY – What WE SAY

THEY SAY we face a CWD crisis that jeopardizes Wisconsin's $1,000,000,000 deer hunting industry.

WE SAY the crisis is of their own making. Their unfounded panic, hysteria and hype is what led to the 2002 drop in license sales and deer kill. Their on-going and unpopular tactics are only making matters worse. The industry is threatened more by CWD policy than by CWD itself. Hunters in other states have shown widespread willingness to live with CWD in the deer/elk herd. Wisconsin hunters did the same last fall.

THEY SAY we should enjoy the added hunting opportunity.

WE SAY this is slaughter, not hunting. DNR enthusiasm about expanded hunting opportunities (such as our chance to collect a set of antlers in velvet) insulted true hunters. It revealed a fundamental misunderstanding of what it means to hunt on the part of the very agency that is supposed to be promoting hunting. Both hunters and non-hunters lost respect for those who killed piles of deer for the dumpster. Many who participated in this wasteful killing, and some who only witnessed the shooting, may never again be able to find any joy in hunting.

THEY SAY that there is no reasonable alternative to eradication.

WE SAY there are several options, none of which was openly discussed with landowners to see what was most acceptable and, therefore, achievable. The option we prefer is disease control and research, not eradication. This is being done in all other states where CWD has been found. Only Wisconsin has chosen eradication.

THEY SAY additional killing tools and extreme measures are needed because landowners have not been able to do the killing themselves.

WE SAY the reason they fell short of their "total kill" goal is not due to an inability, but an unwillingness, on the part of landowners. We do not need more tools or government help. We need a CWD policy that makes more sense.

THEY SAY the eradication process is a minor nuisance for landowners.

WE SAY it has thoroughly disrupted our lives, violated our property rights, and caused us financial damage. It is not acceptable to have a continuous series of killing periods and blaze orange rules. After heaping scorn on poachers for years, DNR sharpshooters have now adopted their ways (night shooting, shooting from vehicles off roads, trespassing, wasting meat). This intrusive CWD policy has greatly harmed our rural life style.

THEY SAY that CWD policy is written in stone and can not be changed.

WE SAY that directly contradicts the principles of adaptive management that the DNR says it is following. State policy does change all the time – that is why new public servants are elected/reelected/cast aside from time to time. But it does take vision, skill, courage and effort to change flawed policy. This is what we are asking for now. Somebody needs to show real leadership and step up to this challenge.

THEY SAY there is no hope that genetic resistance will take care of CWD naturally.

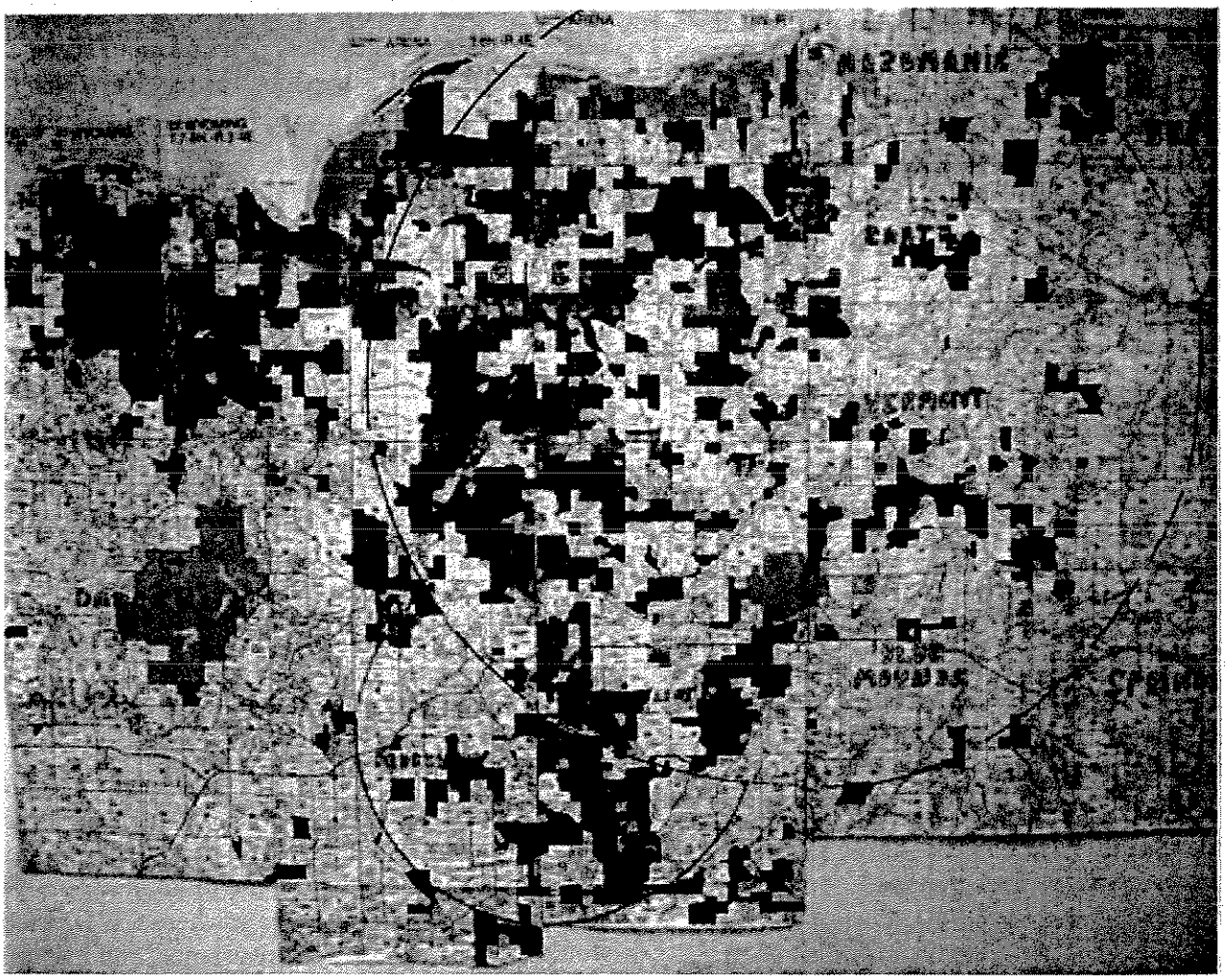
WE SAY this is a highly unlikely contention, since that is the mechanism that animals use to counter other diseases. But time is needed for this solution to play out. Furthermore, killing all deer in an area where CWD is found would eliminate those with resistance, and may actually prevent or delay a genetic resistance solution.

How Bad Can It Get?

The DNR has for months been discussing the pros and cons of using a bounty incentive to kill deer remaining in the eradication zone. That would pit neighbor against neighbor, and poacher against landowner.

But, it gets worse! The author of the DNR's eradication model has written: "When landowner cooperation is not forthcoming and deer persist on their property, WDNR asserts eminent domain, removes deer, and bills landowner."

Red Petition Map: The colored parcels represent the holdings of landowners who have signed the petition opposing the DNR's current "total kill" CWD policy. We invite you to add your property to this protest map by signing and mailing the Landowner's Petition found in this newsletter.



Wild deer eradication is considered "unattainable in . . . endemic CWD situations".

Professor Elizabeth S. Williams, et. al.

"Chronic Wasting Disease in Mule Deer and Elk: A Review with Recommendations for Management." Journal of Wildlife Management, July, 2002 66(3): 551-563

"The strategy has failed. If the animal rights folks came to me and said, 'Can you design a program that would have the biggest possible negative effect on hunting?' I couldn't have put together anything this creative and this clever."

Dr. Thomas A. Heberlein, Professor Emeritus

Department of Rural Sociology, UW—Madison

The Capitol Times, Thursday, February 27, 2003, p. 9A

"Eradication of CWD is not a feasible goal in wild populations."

Dr. Charles H. Southwick, Professor Emeritus

Environmental and Organismic Biology, University of Colorado

Testimony to the U.S. house of Representatives Committee on Resources, 2002.

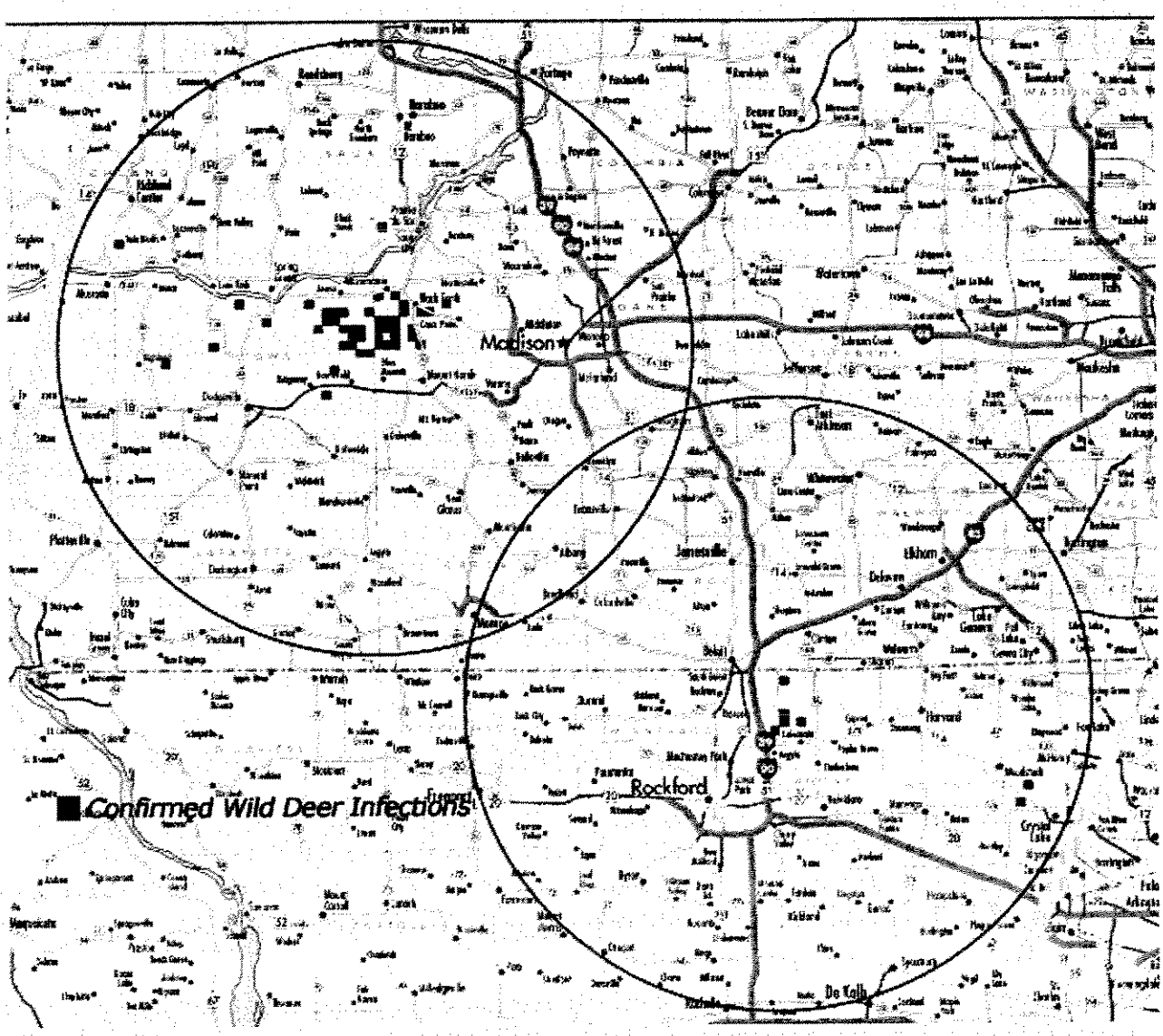
"My strong opinion is that CWD and other diseases never will be eradicated from wild deer herds. They just become a factor to condsider in management."

Professor James C. Kroll, Founder and Director

The Institute for White-tailed Deer Management and Research

Steven F. Austin State University, Nacogdoches, TX

North American Whitetail, 21(8): 20-27.2002



This map shows where CWD had been found in the wild deer herd during the past year. The area of each red circle is over 4,000 miles squared. Other locations determined by a private laboratory are being contested by the DNR. In addition, game farms in Walworth, Portage and Manitowoc counties have had animals test positive for CWD.

Wisconsin's Doomsday CWD Model

In April (2002) the DNR invited John Cary, a staff member in the UW-Madison Dept. of Wildlife Ecology to develop a numerical model that would simulate the spread of CWD in Wisconsin. He spent many hours trying to decide what things (variables) mattered, how much they mattered (input values), and how they should be linked (add, subtract, multiply, divide) together. From these choices he built a complex computer model based on five sub-models. Since facts about CWD are scarce, and little was known about the distribution or prevalence of CWD in Wisconsin, many assumptions had to be made. Eventually his creation was used to develop a slide show illustrating how CWD could cause a rapid and total collapse of the deer herd.

The model is an interesting analytical and predictive tool. It let Cary compare what might happen if he did this, or did that, or did nothing with respect to each of the input values in his five sub-models. The resulting model is best viewed as an ongoing experiment, an interesting and potentially insightful "work in progress." But its forecasting ability has yet to be tested against reality on the ground.

Cary provided an honest appraisal of his model by stating "DISCLAIMER: modeling results should be used with caution." He recognized that "each input value is a valid research project (and likely point of contention," and that "the structure of the submodels themselves is debatable – CWD transmission especially." He further cautioned that the "best available data are not necessarily accurate or appropriate for southern Wisconsin," the "sensitivity [of the model] needs to be explored," and deer "dispersal is a particularly sensitive parameter which is poorly understood for southern Wisconsin."

This all makes for a highly speculative and cautionary modeling statement. Unfortunately, the DNR chose to promote this preliminary and highly qualified model as justification for its quick, forceful and expensive CWD policy. We think this action was premature until much more is known about CWD in general, and until we have a much better grasp of the distribution and prevalence of the disease in Wisconsin.

DNR "kills" 13,000 deer in Eradication Zone with "stroke of pen."

In the winter of 2002 the DNR folks in charge of CWD matters announced that the "total kill" zone contained 25,000 - 30,000 deer. At the same time the DNR produced its "2001-2002 Overwinter Deer Density Estimates Compared to Overwinter Population Goals" data based on sex-age-kill figures, which showed that approximately 15,000 deer resided in the Eradication Zone. After conducting an aerial survey in March 2003, the DNR cut its 30,000 estimate by nearly 50%! They now say there were probably between 16,400 and 17,900 deer in the "total kill" zone in the fall of 2002. In other words, the newly "revised" total nearly matches the overwinter density estimate.

So why was the overwinter estimate inflated by nearly 100% in claiming 30,000 deer must be killed? What purposes were served by this knowing distortion? Your guess is as good as ours.

What does the statewide CWD surveillance testing tell us"?

The Cary model assumes a single deer caused the CWD outbreak in Wisconsin. By this logic, one diseased deer could plant the seeds of CWD at any place in the state at any time. And that implies a negative test from every deer in the state is needed before we can feel truly safe from the disease. The same would be needed for every penned deer/elk in the state. Of course, this would require killing every animal! This is clearly impractical.

In this regard, results from the current statewide surveillance program may be encouraging, but they are by no means definitive. Far greater numbers of older deer will have to be tested in each county before the rest of the state can be declared "free of CWD" with any real confidence. Given the 18 month minimum latency/gestation period before CWD can be detected in deer/elk, meaningful surveillance will require many years of extensive testing.

Where and when CWD has been found in North America

Finding CWD was extremely difficult before 1996 when new testing methods became available. The new test, along with the greatly expanded national surveillance testing that followed, largely explains the recent dramatic rise in states reporting the disease.

Wild deer/elk population:

Colorado (1981), Wyoming (mid-1980's), Saskatchewan (2000), Nebraska (2001), South Dakota (2002), Wisconsin (2002), Illinois (2002), New Mexico (2002), Utah (2003), Alberta (2003)

Game Farms, Shooting Preserves and Deer/Elk Research Facilities:

Colorado (detected 1967; diagnosed 1977), Wyoming (late 1970's), Saskatchewan (1996), South Dakota (1997), Nebraska (1998), Oklahoma (1998), Montana (1999), Kansas (2001), Alberta (2002), Minnesota (2002), Wisconsin (2002)

Important note: CWD has been discovered in over 40 new game management units nationally since it was reported in Wisconsin last February (2002).

---------- Clip and mail to CALFARR, P.O. Box 148, Arena, WI 53503----------

Landowners' Petition

As land owners in or near the CWD Eradication Zone, we agree that recent deer density is not sustainable and should be reduced to a level that better balances various economic, political and environmental concerns. But we also feel that DNR CWD policy calls for an unjustified, unfair and unreasonable sacrifice from landowners in our part of the state. Thus, we, the undersigned, call on the elected leaders and DNR employees of Wisconsin to postpone the planned eradication of the deer herd in our area until enough is known about CWD to devise a more acceptable plan. Before further pursuing the "total kill" policy we propose that:

- The DNR test all deer killed in the Herd Reduction Zone for surveillance purposes.
- A statistically meaningful, multi-year testing effort be made to determine the true distribution and prevalence rate of CWD statewide.
- More research be done on CWD, particularly with respect to cause, incubation, transmission and dispersal.
- All possible effort be made to make available to hunters a fast, accurate, convenient and inexpensive CWD test so that people will not avoid hunting or needlessly waste venison.
- Alternatives to the "total kill" policy be debated and voted on in a public forum.
- Government shooting activity be stopped except for culling sick animals and exploratory surveillance.

Signature: _____ Acres Owned: _____

Address: _____ Township: _____

Phone: _____ Section(s): _____

Signature: _____ Acres Owned: _____

Address: _____ Township: _____

Phone: _____ Section(s): _____

We need your help

- Fill out petition on page 7 (send to CALFARR address)
- Contribute what you can (send to CALFARR address)
- Volunteer your time and energy (call or write CALFARR)
- Contact your political representatives:
  Dale Schultz 608/266-0703
  sen.schultz@legis.state.wi.us
  Steven Freese 608/266-7502
  rep.freese@legis.state.wi.us
  Jon Erpenbach 608/266-6670
  sen.erpenbach@legis.state.wi.us
  Sondy Pope-Roberts 608/266-3520
  rep.pope-roberts@legis.state.wi.us
  Dave Travis 608/266-5340
  rep.travis@legis.state.wi.us
  Mike Powers 608/266-1192
  rep.powers@legis.state.wi.us
- Let DNR secretary Scott Hassett know how you feel: 608/266-0865 Fax: 608/266-6983
- Send the local DNR board members your views:
  Jonathan P. Ela, 2130 Chamberline Ave., Madison, WI 53726, 608/238-8187
  Howard D. Poulson, 1212 Deming Way, P. O. Box 5550, Madison, WI 53705, 608/828-5700

Landowner CWD Newsletter

Welcome to the first issue of a newsletter on Wisconsin Chronic Wasting Disease policy produced and distributed by Citizens and Landowners for a Rational Response (CALFARR). We are a diverse group of hunters, landowners, and citizens united with a common cause to stop the DNR's "total kill" CWD plan. We are also seeking to participate actively in the formulation of scientifically sound, cooperative and adaptive deer management practices in southwest Wisconsin.

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(60 75 90
CALFARR

Good morning.

I am a member of Citizens and Land Owners for a Rational Response. My name is John Forseth. I was born and raised in Arena Township. I graduated from Arena High School, left home for college and was out of the area for 25 years and then in 1983 moved back. This fall will be my 50th year of deer hunting on the home farm.

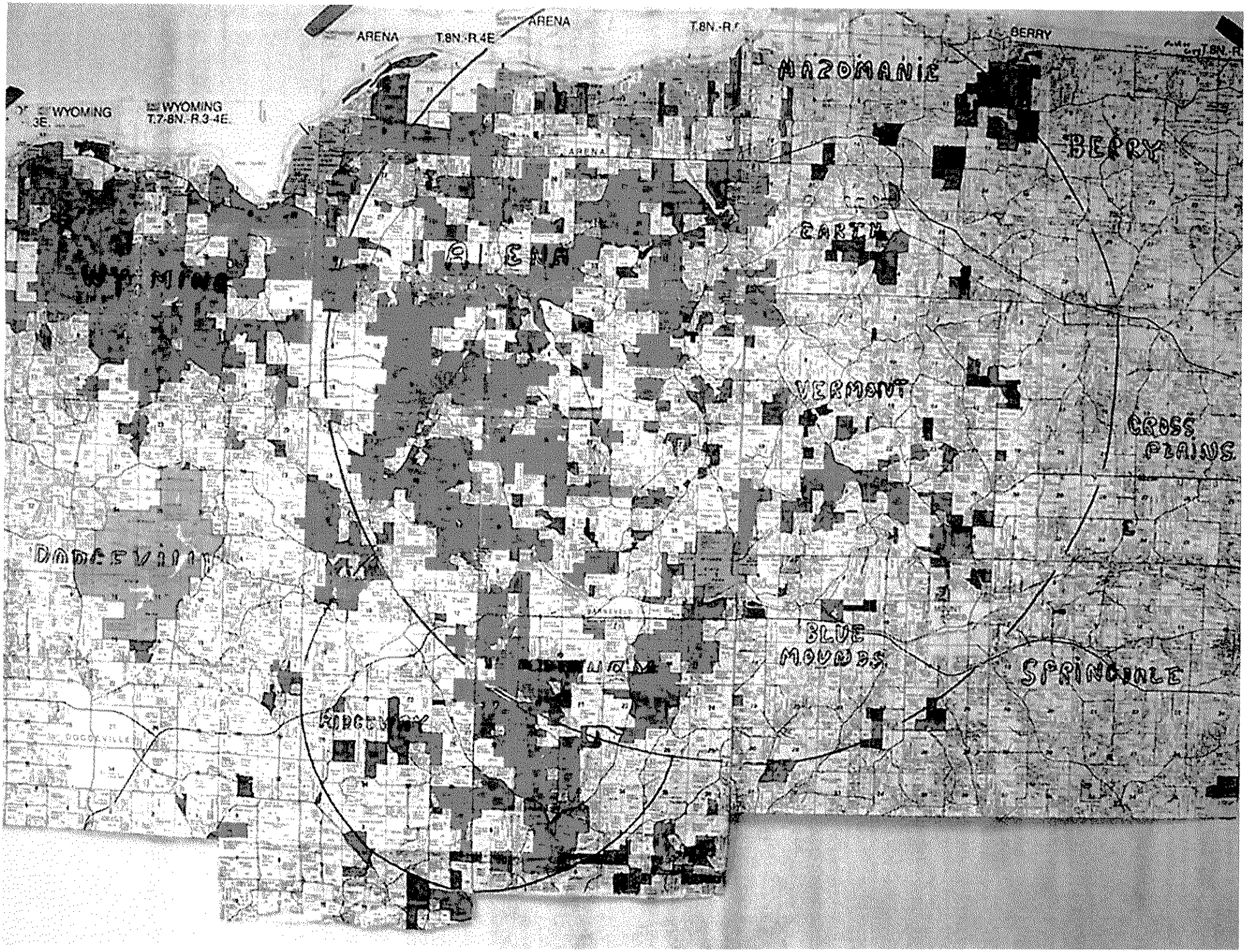
I am speaking in opposition to the permanent rule change. I, along with almost everyone, was shocked when the DNR announced their deer eradication plan as their means to get rid of CWD. Once it sunk in that they were serious, some of us landowners started to discuss and research the best we could, using the internet and whatever other resources we could find to put and end to this craziness of killing all the deer even if it meant shooting from helicopters. Despite the fact that the DNR was saying that they had overwhelming landowner support, we, in fact, were finding just the opposite.

We then organized and held our own meeting, discussed our options and basically cut it down to two. One was to pursue legal avenues to have the eradication plan stopped and the second was to circulate a petition (handout #1) to be signed by landowners in or near the eradication zone. We opted for the petition drive and with a handful of hard working volunteers, signed nearly 90,000 acres to our petition. The petition signers' land is colored in red (handout #2) on the handout and the map I am holding up. Our research shows that eight out of ten people that we found at home would sign the petition.

Our efforts were concentrated mainly on the Iowa County side of the map because that is where most of our group lives and owns land. Needless to say, there is not overwhelming landowner support for the DNR's plan. In fact, the petition drive was held before there was much written or known about the eradication. The sentiment among landowners against eradication runs much higher today and by the DNR's own findings, the CWD plan will not work without landowner participation.

We have held meetings with Secretary Bazell and Secretary Hassett and really felt like we were not taken seriously. We have evolved into a very

good, dedicated group, which includes people from ages twenty-seven to eighty, with professions of farmers, professors, salaried workers, retirees, hourly employees and business owners. We need to be recognized by the DNR and to become partners in the CWD battle if there is to be any chance of success.


ARENA
T.8N.-R.4E.
ARENA
T.8N.-R.
BERRY
WYOMING
T.7-8N.-R.3-4E.
WYOMING
MAZOMANIE
BERRY
EARTH
VERMONT
CROSS PLAINS
BLUE MOUNDS
SPRINGDALE

Opposition Arguments to the EIS and Bills CH03-016 and CH03-017 to Attempt Eradication of CWD from Wisconsin's White-tailed Deer Herd

Anthony C. Grabski, Ph.D.
Blue Mounds, WI
May 14, 2003

Good morning and thank you for this opportunity to present my views on the proposed bills to control CWD in Wisconsin. My name is Anthony Grabski, and I hold both MS and Ph.D. degrees in Bacteriology from the University of Wisconsin-Madison. I am currently employed as a Protein Biochemist, I am a hunter, and I own 48 acres near the center of the proposed eradication zone. Although I represent myself I am a member of Citizens and Landowners for a Rational Response to CWD (CALFARR) and my views are shared by hundreds of concerned citizens both within and outside of the CWD eradication zone with whom I have discussed these issues.

A little over a year after CWD was detected in Wisconsin we find ourselves in the midst of a "CWD mess" as summarized by Professor Scott Craven, Chairman of the University of Wisconsin Department of Wildlife Ecology. This CWD mess involves not only the complex biological balance of a little known disease affecting deer in their environment, but also many social, financial, and political elements. History sometimes repeats itself, and in 1937 an outbreak of cattle tick fever prompted Florida Wildlife Officials to embark on a deer eradication program based on the assumption that deer were carriers of the tick, and therefore, the disease. A prominent game manager was contacted by the Executive Director of the Audubon Society to assess the situation, and the game manager summarized the eradication effort as follows:

> **This episode shows that a scientific bureau, confronted with a question of wildlife eradication, may prefer to lose the wildlife than to lose time in scientific research for alternatives. In one sense this is water over the dam, but in another it carries a lesson for the future. At best, the scientific base in this case was sketchy, and no imagination had been used in searching for less destructive alternatives.**
>
> **It is time for us to learn caution and restraint in our power to eradicate wild things.**
> **--Aldo Leopold, December 5th, 1944**

The proposed rules to eradicate CWD from our State are based on similar 'sketchy science'. If these rules become law they will only succeed in eradicating deer and deer hunting, but not CWD. We have listened for over a year as DNR officials stated that the eradication is based on "the best available science". I too have independently researched "the best available science" and have reached the opposite conclusion. Random deer eradication from nearly 1,000 square miles of hilly, wooded, private property, as a means to eliminate CWD is not only irrational and impractical, but also impossible. Conclusions from the scientific literature support this view.

2000, *Journal of Wildlife Diseases*

> Effective strategies for controlling or eliminating CWD in wild deer and elk have not been identified. It follows that random culling via harvest or other means may be relatively ineffective in reducing CWD prevalence.

2001, *Journal of Wildlife Management*

> Once introduced and established , most infectious diseases are extremely difficult to eliminate from free-ranging populations. It follows that a new or emerging wildlife disease should be carefully evaluated early on to assess both its potential importance and prospects for effective management. The biological mechanisms underlying CWD transmission are poorly understood, and as a result model mechanisms are at best a collection of educated guesses.

How are landowners expected to cooperate in an obviously impossible eradication effort when Dr. Elizabeth Williams' own publications state effective strategies for controlling or eliminating CWD have not been identified and random culling is ineffective? Deer (disease) eradication from wild deer in an 874 square mile area is even more ridiculous now than the originally ridiculous 411 square mile zone. I use the word ridiculous to describe the plan, because I am at a loss for a better word to describe this childish notion.

Finally, I strongly disagree with comparison of the Sandhill deer removal hunts of 1972 as a equitable example of successful eradication. Sandhill was a nine square mile fenced enclosure, containing approximately 500 deer, with total landowner cooperation. The Sandhill removal hunt took considerable time and effort even at this relatively small scale. The new eradication zone is at least 874 square miles (and it will certainly expand), 25 to 50,000 animals must be removed, and landowners will not participate in such lunacy. The current solution to shoot their way out of this multifaceted problem is far too simple. Killing tens of thousands of deer for the dumpster and reducing the deer to toxic waste unfit for landfills is an insult to human intelligence and a disgrace to the white-tailed deer and our deer hunting heritage. I urge you to vote against CH03-016 and CH03-017 and request a more practical, imaginative, and less destructive alternative.

Thank your for you consideration.

Anthony C. Grabski
5180 Ridge View Road
Blue Mounds, WI 53517